J. Pascual

*Hospital Universitario Marqués de Valdecilla e IDIVAL, Santander, Spain*

*E-mail address:* juliopascual@telefonica.net

2173-5808/


# Reversible subacute chorea caused by vitamin B12 deficiency☆

## Corea subagudo reversible por déficit de vitamina B12

*Dear Editor,*

We present the case of a 47-year-old man who worked as a baker and was admitted to hospital due a 4-day history of subacute symptoms of choreoathetosis predominantly affecting the head and left limbs, as well as motor impersistence and dysarthria. The physical, neuropsychological, and psychiatric examinations revealed no other relevant findings, including normal muscle balance and polymodal sensitivity. He had no family history of interest and a personal history of migraine, smoking, and moderate alcohol dependence (3 mixed drinks per day; his alcohol consumption habits had not changed in the past few months).

He underwent a complete blood study (electrolytes; TSH; PTH; copper; ceruloplasmin; folate; and vitamins E, $B_1$, and $B_{12}$), autoimmune tests (ANA; ENA; ANCA; TPO; lupus anticoagulants; and cardiolipin, gliadin, and intrinsic factor antibodies), serology tests, blood smear, test for antineuronal antibodies, lumbar puncture, and a urine toxicology test. The only relevant findings were macrocytosis (MCV = 115 fL [normal values: 80—100 fL]) and vitamin $B_{12}$ deficiency (148 pg/mL [normal values: 200—900 pg/mL]). Our patient displayed no anaemia and no other imbalances. A brain MRI scan revealed a slightly hyperintense area adjacent to the third ventricle and in the mammillary bodies, which may be related to our patient's alcohol consumption habits; no other alterations were found (Fig. 1). A CT scan of the neck, chest, abdomen, and pelvis showed no signs of hidden neoplasm.

While waiting for results from the laboratory tests, we initiated empirical treatment with IV immunoglobulins, which did not improve our patient's symptoms. We ruled out toxic, infectious, dysimmune, vascular, neoplastic, and paraneoplastic causes. Since we suspected subacute choreoathetosis due to vitamin $B_{12}$ deficiency, the patient started IM vitamin $B_{12}$ (1000 μg/day for 7 days, followed by 1000 μg/week for a month, and 1000 μg/month) plus oral tiapride dosed at 50 mg every 8 hours; symptoms improved steadily and had resolved completely one week after treatment onset. Our patient remained asymptomatic one month later, so tiapride was discontinued. At 3 months, he continued to be asymptomatic and showed normal vitamin $B_{12}$ levels.

Vitamin $B_{12}$ deficiency is typically associated with such neurological disorders as subacute combined degeneration, dementia, and polyneuropathy.[1] However, the literature also includes isolated cases of such extrapyramidal disorders as parkinsonism, focal dystonia, ataxia, myoclonus, or even chorea associated with vitamin $B_{12}$ deficiency.[1—6]

From a clinical viewpoint, chorea secondary to vitamin $B_{12}$ deficiency may be either unilateral or markedly asymmetrical with no associated structural lesions, as with other types of chorea (those linked to deficiencies or metabolic changes).[3] Chorea linked to vitamin $B_{12}$ deficiency is thought to be caused by the glutamatergic activation of the basal ganglia due to excess of homocysteine (an NMDA agonist); in addition, there may be a potential neurotoxic effect resulting from an excess of methyltetrahydrofolate (an agonist of kainic acid which induces Huntington chorea in animal models).[2,7,8] Unfortunately, homocysteine levels in our patient were not tested during the acute phase. If sensory alterations are associated with vitamin $B_{12}$ deficiency,[3] they may contribute to choreoathetosis by a deafferentation mechanism. However, this does not seem to be a necessary

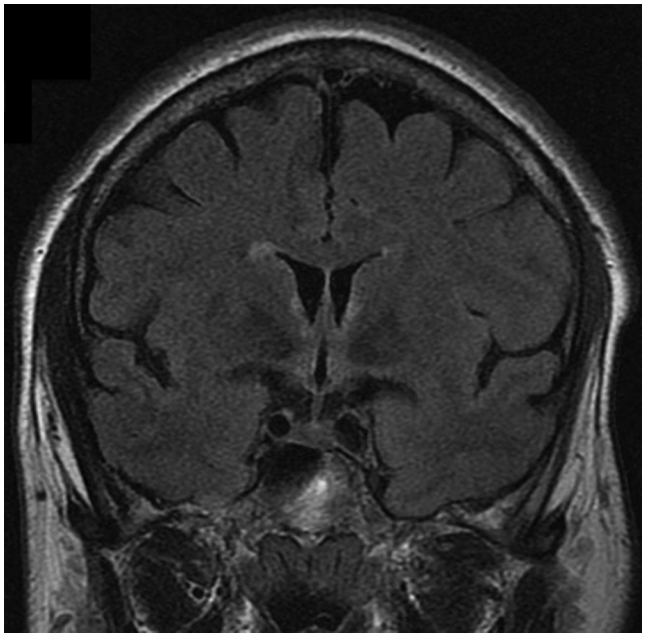

**Figure 1** Brain MRI scan (coronal FLAIR sequence) showing hyperintensities adjacent to the third ventricle and in the mammillary bodies.

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condition; in our patient and in other cases, examinations revealed no sensory alterations.[2,6]

As in other cases in the literature, our patient's diagnosis of subacute reversible chorea caused by vitamin $B_{12}$ deficiency was based on the temporal relation between symptoms and vitamin $B_{12}$ deficiency, the exclusion of other potential causes of chorea, symptom resolution having coincided with stabilisation of vitamin $B_{12}$ levels, and the lack of recurrence after tiapride was discontinued. Given that not all patients with low vitamin $B_{12}$ levels develop neurological symptoms, other factors such as long-term alcohol abuse are likely to trigger the syndrome. MRI findings in our patient match those classically associated with Wernicke encephalopathy due to vitamin $B_1$ deficiency[9]; however, our patient showed normal vitamin $B_1$ levels.

In conclusion, chorea is a rare manifestation of vitamin $B_{12}$ deficiency which can be resolved with replacement therapy. We recommend including vitamin $B_{12}$ tests in the aetiological study of chorea of unknown origin.

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## Conflicts of interest

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F. Gascón-Giménez*, F. Coret-Ferrer

*Servicio de Neurología, Hospital Clínico Universitario, Valencia, Spain*

* Corresponding author.
*E-mail address:* fgascongimenez@gmail.com
(F. Gascón-Giménez).
2173-5808/