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Differences between chronic migraine with and without medication overuse☆

Diferencias entre migraña crónica con y sin uso excesivo de medicación

Dear Editor:

I read with great interest the outstanding article by Rojo et al.[1] analysing the differences between patients with chronic migraine (CM) with and without medication overuse (MO). According to this study, only 57% of these patients had received preventive treatment with the most frequent type being antidepressants; most patients had received only one type of preventive treatment. These findings should encourage self-criticism among general practitioners and neurologists alike regarding the way we manage migraine. This entity constitutes the main reason for neurology consultations in Spain, and it has been ranked eighth by the WHO in terms of impact on activities of daily living.[2]

Except for longer progression times, Rojo et al. found no differences between these 2 subgroups of patients with CM. This suggests that: (1) CM with and without MO are not separate entities, and (2) in most cases, MO is the consequence of CM, rather than the cause. Departing from the conventional wisdom that the vast majority of patients with CM meet criteria for MO, the above study reports that just over half of the patients with CM (258 out of 434, that is, 59.5%, not 72.2% as cited in the abstract [a considerable percentage by any standards]) met the criteria for MO, confirming once more that MO is not a necessary condition for migraine chronification.

Conflicts of interest

The author has no conflicts of interest to declare.

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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

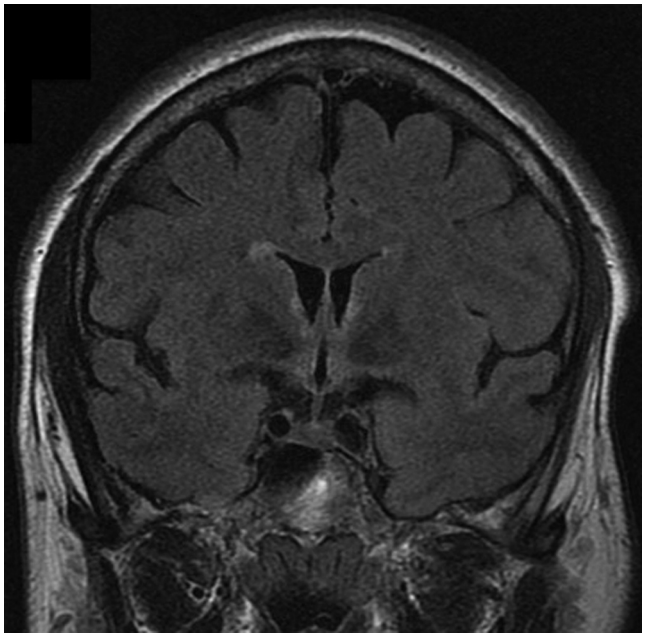

Figure 1 Brain MRI scan (coronal FLAIR sequence) showing hyperintensities adjacent to the third ventricle and in the mammillary bodies.

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