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Case Report

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Hypomagnesemia-induced cerebellar syndrome: a case report

Abstract

Magnesium is the second most abundant intracellular cation after potassium and is involved in over 600 enzymatic reactions that are essential for life. Hypomagnesemia (serum magnesium concentration <1.8 mg/dL (< 0.70 mmol/L)), is longstanding known to cause many clinical disorders: other electrolyte abnormalities, life-threatening arrhythmias and various neurological manifestations, from muscle cramps and myopathy, to vertigo, nystagmus, depression, acute confusional state and seizures. In the last few years some case reports have highlighted the possible existence of a peculiar hypomagnesemia induced cerebellar syndrome (HiCS). Here we present a clinical case of a 74-years-old man with severe hypomagnesemia presenting with vomiting, gait instability, diffuse tremor, associated with neuro-otological signs of cerebellar dysfunction and a MRI hyperintense lesion at cerebellar nodulus with clinico-radiological resolution after magnesium repletion.

Keywords: magnesium, hypomagnesemia, cerebellum, dizziness, ataxia, head impulse test

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Introduction

Hypomagnesemia-induced cerebellar syndrome (HiCS) has been recently hypothesized to be a distinct disease entity due to remarkable similarities of clinical, laboratory, and Magnetic Resonance Imaging (MRI) features reported in medical literature.[1] Here we present the case of a 74-years-old man with severe hypomagnesemia[2] with a subacute onset cerebellar syndrome discussing his clinical, laboratory and MRI characteristics. A written informed consent was signed by the patient.

Case report

A 74-years-old white man presented at the Emergency Department (ED) of Careggi Hospital in Florence (Italy) in November 2021 because of nausea and postprandial vomiting episodes reported in the previous 30 days determining progressive weight loss, associated with dizziness, worsening gait impairment until having difficulties to get out of bed and diffuse tremor.

He is an ex-worker, now retired. He lives with his wife. Pre-existing conditions were chronic obstructive pulmonary disease associated with severe smoking habit, arterial hypertension, diabetes, dyslipidemia, chronic obliterative arterial disease of the lower limbs and a history of heavy alcohol consumption, which was interrupted one month earlier; he was on the following medications: amlodipine, valsartan, clopidogrel, simvastatine, metformine, lansoprazole, edoxaban, liraglutide, umeclidine, vilanterole, fluticasone.

In the ED he presented with gait instability, diffuse tremor, rigidity and dysarthria. During ED observation, he developed vertigo, followed by an acute confusional state requiring sedation with midazolam and diazepam. Vital signs were in normal range, he was apyretic; a head computed tomography (CT) was performed, showing signs of moderate leukoaraiosis. In suspect of Wernicke's encephalopathy (WE) treatment with intravenous thiamine was started. A brain MRI was performed and no ischemic lesions were found, other than chronic microangiopathic lesions in the white matter of both cerebral hemispheres. However, a subtle hyperintensity in Fluid Attenuated Inversion Recovery (FLAIR) (Figure 1), with a dubious diffusion weighted imaging (DWI) (Figure 2) signal hyperintensity at the cerebellar nodulus was found; apparent diffusion coefficient (ADC) sequence was negative (not shown).

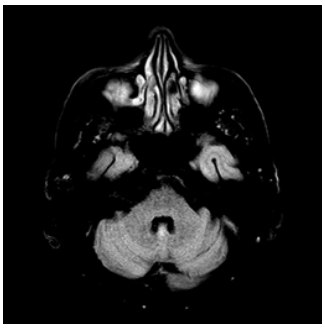

Figure 1 In axial FLAIR-T2 sequence a subtle hyperintensity at the cerebellar nodulus is visible.

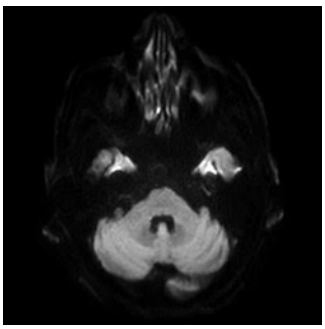

Figure 2 In DWI sequence a dubious hyperintensity at the cerebellar nodulus is visible.

After the acute confusional episode was resolved, an audiological examination was carried out. Under Frenzel's goggles, a persistent down-beating nystagmus of small amplitude, was observed in supine position and on right side lateral head position; this nystagmus, while maintaining the same temporal characteristics, appeared more evident in Rose position and both in right and left Dix-Hallpike's positionings; conversely, on left side lateral head position there was a small and persistent apogeotropic right-beating nystagmus. When the patient was brought in sitting position, there was a tendency to truncal retropulsion, with the same small and persistent down-beating nystagmus, which was of 1st and 2nd degree, and was only slightly reinforced in lateral and downward gaze, but became transitorily stronger after head shaking. Noteworthy, bedside horizontal Head Impulse Test (HIT) elicited clear reversed, anti-compensatory, saccades on both sides. Cover test and vestibulo-ocular reflex (VOR) cancellation were normal. Finally, our patient showed Babinski's asynergia and truncal ataxia in sitting position and was not able to maintain the standing position without support (that is grade 3 of Carmona's ataxia). Thus, all oto-neurological signs converged to a predominantly cerebellar dysfunction.

The patient was hospitalized, underwent angio-CT of neck and head vessels and a total-body CT, which came back negative. Laboratory analysis displayed severe hypomagnesemia (0,5mg/dL). Therefore, intravenous magnesium was administered with normalization of serum magnesium levels. He was discharged at home persisting only mild ataxic gait. An oral supplementation of magnesium was recommended. Twenty days later he went back at the ED complaining a recrudescence of his symptoms, in particular vomiting, dizziness and progressively worsening gait ataxia. Laboratory analysis revealed very low magnesium levels (0,3mg/dL). After intravenous magnesium supplementation neurological signs and symptoms got rapidly better. Head MRI didn't show cerebellar hyperintensities and the audiological evaluation found only mild signs still suggestive of central vestibular dysfunction of the cerebellar type, but the reversed saccades at the HIT were no longer detectable. After gastroenterological consultation, therapy with lansoprazole was discontinued as a potential extrarenal cause of hypomagnesemia. The patient was discharged at home. At the last neurological and audiological evaluation the patient displayed a normal examination with the exception of a cogwheel smooth pursuit on the horizontal plane when looking to the right.

Discussion

This case-report highlights the importance of a complete oto-neurological evaluation in order to properly localize the site of neurological damage, and orient the clinical reasoning on differential diagnosis.

The bedside HIT is a useful method to quickly evaluate the vestibular function in patients with dizziness and or vertigo. Specifically, HIT can identify peripheral vestibular hypofunction by detecting corrective saccades in the opposite direction of head rotation, that is compensatory saccades, due to decreased VOR gain. Conversely, in cerebellar disorders HIT is mostly normal, but horizontal (compensatory) or downward (perverted) corrective saccades have been described during horizontal HIT.[3,4] Instead, our patient showed reversed (paradoxical) corrective saccades in the same direction of head rotation during bedside HIT. This paradoxical response should be considered an additional bedside cerebellar sign and may be useful in detecting cerebellar dysfunction.[5]

Indeed, the vestibulo-cerebellum controls the VOR gain through inhibitory Purkinje cell fibers projecting to the vestibular nuclei; therefore, disinhibited vestibular nuclei due to cerebellar lesions may result in increased (excessive) VOR gain and reversed (paradoxical) corrective saccades during HIT.[6] We could infer two structures as neural substrates responsible for the paradoxical HIT: flocculus/paraflocculus because of spontaneous down-beating nystagmus, increasing on lateral and downward gaze; nodulus/uvula because of positional down-beating nystagmus and perverted head-shaking nystagmus. Moreover, truncal ataxia in sitting position and severe postural ataxia in standing position also pointed towards a cerebellar dysfunction.[7] Finally, Babinski's asynergy sign is particularly useful as it allows to evaluate the patient while in bed and in supine position.

In our clinical reasoning we could exclude an ischemic etiology because it couldn't explain the whole clinical spectrum, the subacute progression of symptoms and the atypical MRI cerebellar lesion; WE could be excluded because the patient hadn't an encephalopathy, neuro-ophthalmological signs were not characteristics of WE and MRI didn't disclose typical bilateral mammillary bodies lesions.[8] Conversely, severe hypomagnesemia could explain the whole clinical picture, the MRI hyperintensity of cerebellar nodulus, the dramatic improvement of symptoms after normalization of magnesium levels; their recrudescence after recurrence of hypomagnesemia was quite confirmatory.

Mg2+ deficiency promotes oxidative stress and inflammation in endothelial cells, determining capillary leakage and BBB disruption.[9] The relatively lack of sympathetic innervation in the posterior circulation is the likely mechanism for the preferential involvement of the posterior part of the brain and for mimicking MRI lesions of posterior reversible encephalopathy syndrome (PRES). However, the cause seems different predominantly being hypomagnesemia in HiCS and hypertension in PRES. Hypomagnesemia in our patient has been attributed to chronic consumption of PPIs;[10] this association has been observed in particular after more than one year of drug consumption, especially in elderly.[11,12]

Conclusion

In conclusion this case report strengthens the idea that hypomagnesemia-induced cerebellar syndrome could be a distinct disease entity. ED physicians and neurologists should be aware of this condition, since serum magnesium level measurement is not part of the routine electrolyte panel and since the prompt treatment of severe hypomagnesemia can avoid potentially life-threatening conditions such as ventricular arrhythmias and seizures.

Acknowledgments

None.

Conflicts of interest

Authors declare that there is no conflict of interest.

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