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POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME RELATED TO SEVERE HYPOMAGNESAEMIA

Martín Naya Rosato[1], María Luisa Núñez Calvo[1], Lucía Barrera López[1,2]

[1] Internal Medicine Department, Hospital Clínico Universitario de Santiago de Compostela, Santiago de Compostela, Spain
[2] Fundación Pública Galega, Instituto de Investigación Sanitaria de Santiago de Compostela, Spain

Corresponding author's e-mail: Martin.Naya.Rosato@sergas.es

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ABSTRACT

Introduction: Posterior reversible encephalopathy syndrome (PRES) might be associated with a wide spectrum of symptoms, including irreversible neurological damage. Management is primarily symptomatic and focuses on addressing the underlying causes.

Case description: A 65-year-old female with a medical history of smoking, arterial hypertension and dyslipidaemia, was admitted due to dysarthria associated with ataxia, visual disturbances and diarrhoea. The neurological physical examination revealed a mild decreased strength in both legs (4/5), mild dysdiadochokinaesia and mild heel-knee dysmetria. Remarkable laboratory findings showed potassium 2.6 mmol/l and magnesium 0.24 mg/dl. Brain computed tomography (CT) showed no suggestive ischaemic areas. During hospitalisation, potassium and magnesium levels were initially corrected through intravenous supplementation. A brain magnetic resonance imaging (MRI) scan revealed symmetrical cortical alterations in the posterior regions of both cerebellar hemispheres, consistent with a diagnosis of PRES. After the exclusion of other potentially related diseases, the final diagnosis was PRES in the setting of severe hypomagnesaemia.

Discussion: PRES pathophysiology is unknown, while its incidence is probably underestimated. It is suggested that rapid increases in blood pressure are thought to overwhelm the self-regulatory capability of cerebral blood flow, especially in the posterior brain regions where the regulatory flow capacity may be weaker. This might result in increased cerebral perfusion leading to oedema due to capillary leakage. PRES has been associated with poorly controlled arterial hypertension, autoimmune diseases, eclampsia and chemotherapy, among others.

Conclusion: Hypomagnesaemia represents an uncommon but potentially reversible cause of PRES and should be taken into account for differential diagnosis.

KEYWORDS

Dysarthria, hypomagnesaemia, hypokalaemia, posterior reversible encephalopathy syndrome

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LEARNING POINTS

- Chronic drug therapies should always be included in the medical approach.
- The most common aetiology of posterior reversible encephalopathy syndrome is arterial hypertension with poor control and the use of cytotoxic drugs. However, other less common causes, such as severe hypomagnesaemia, should be taken into account.
- The diagnosis of posterior reversible encephalopathy syndrome is based on neuroimaging findings in the correct clinical setting. Therapy is mainly symptomatic and focuses on the underlying causes.

INTRODUCTION

Posterior reversible encephalopathy syndrome (PRES) was first described in 1996[1] in a series of 15 patients presenting with headache, impaired consciousness, seizures and visual disturbances, combined with characteristic neuroimaging findings. Acute and subacute onset is typically and might be associated with a wide spectrum of symptoms, ranging from mild to severe presentation, including irreversible neurological damage[2].

The diagnosis of PRES represents a significant clinical challenge due to its ability to mimic acute vascular pathologies. Diagnosis relies on a high degree of clinical suspicion, supported by acute or subacute neurological symptoms and corresponding findings on neuroimaging[3]. Currently, there is no specific treatment, and management is primarily symptomatic and focuses on addressing the underlying causes[4].

CASE DESCRIPTION

A 65-year-old female patient presented with a medical history of smoking (40 pack-years), arterial hypertension and dyslipidaemia, and was under therapy with ramipril/hydrochlorothiazide 5/25 mg once per day (opd), atorvastatin 20 mg opd and omeprazole 40 mg opd. The patient consulted the Emergency Department one month early for an episode of self-limited dysarthria with no other neurological symptoms, being discharged with a diagnosis of a transient ischaemic attack of undetermined aetiology. Salicylic acid 100 mg opd was indicated. She consulted again with a new episode of dysarthria associated with ataxia and visual disturbances together with diarrhoea, without fever, arthritis or any other systemic symptoms. Physical examination revealed a blood pressure of 153/83 mmHg and a heart rate of 85 beats per minute. Cardiopulmonary auscultation was unremarkable. On neurological examination, she presented a Glasgow Coma Scale of 15/15. Pupils were symmetrical with normal light response, and there were no alterations in the rest of the cranial pair nerves. There was a mild decreased strength in both legs (4/5) with normal sensitivity, and mild dysdiadochokinesia and heel-knee dysmetria were also observed. The rest of the examination was unremarkable. Laboratory tests showed haemoglobin 11.8 g/dl, mean corpuscular volume 83.7 fl, creatinine 0.63 mg/dl, sodium 147 mmol/l, potassium 2.6 mmol/l and magnesium 0.24 mg/dl, with no other electrolyte imbalance. Autoimmunity tests were negative. The electrocardiogram showed sinus rhythm with normal QRS and T waves. Brain computed tomography (CT) showed no suggestive ischaemic areas and the electroencephalogram showed no pathological findings.

During hospitalisation, both hydrochlorothiazide and omeprazole were discontinued due to their potential to induce hypomagnesaemia. Potassium and magnesium levels were initially corrected through intravenous supplementation *(Fig. 1)*. A brain magnetic resonance imaging (MRI) scan revealed symmetrical cortical alterations in the posterior regions of both cerebellar hemispheres *(Fig. 2)*. These findings, which lacked diffusion restriction, were consistent with a diagnosis of PRES. Due to the exclusion of any other potentially related diseases, the final diagnosis was PRES in the setting of severe hypomagnesaemia, most

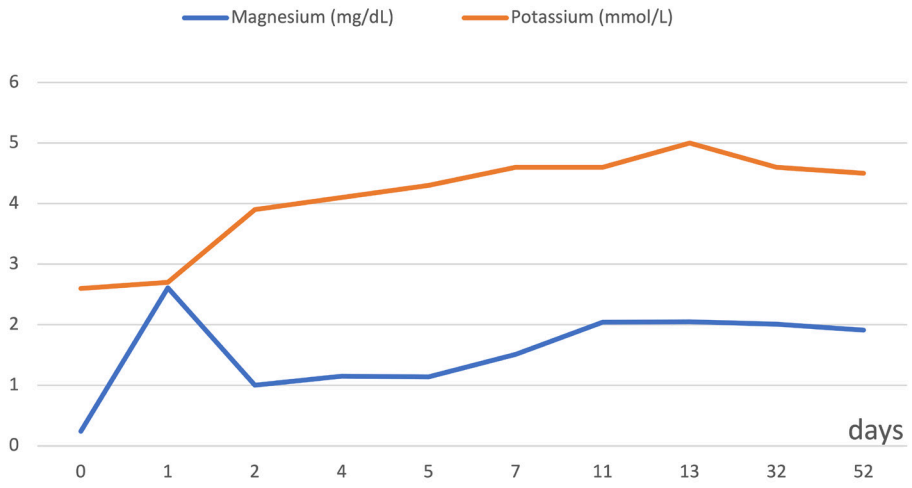


Figure 1. Temporary evolution of magnesium and potassium levels during admission: "day 0" denotes the first day of admission while "day 13" denotes the day of discharge.

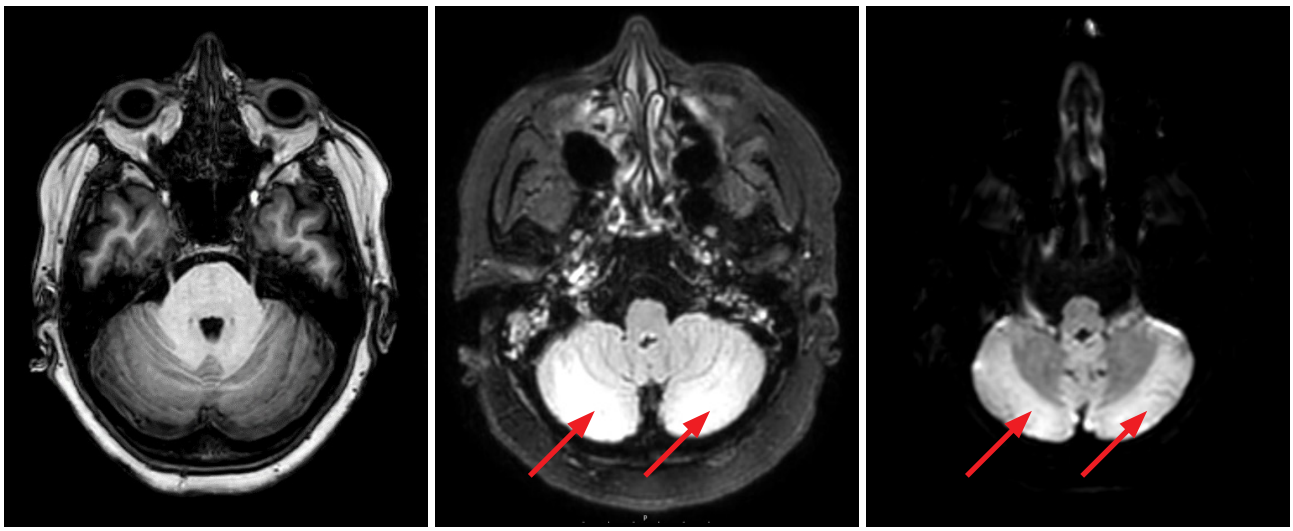


Figure 2. Brain MRI at diagnosis showing cerebellar hyperintensity in flair sequence (arrowhead) and the absence of restriction in diffusion-weighted imaging sequence (arrows).

probably secondary to drug use (omeprazole, thiazides) and digestive losses. The Naranjo score was 4, suggesting that an association between PRES and hypomagnesaemia is possible[5]. After two months of follow-up, the patient presented with complete clinical recovery, with no electrolyte abnormalities *(Fig. 1)*, and resolution of the neuroimage alterations after a brain MRI scan *(Fig. 3)*.

DISCUSSION

Pathophysiology related to PRES is still unknown[3]. On the one hand, rapid increases in blood pressure are thought to overwhelm the self-regulatory capability of cerebral blood flow, especially in the posterior brain regions where it is suggested that the regulatory flow capacity is weaker. This might result in increased cerebral perfusion leading to oedema due to capillary leakage. This hypothesis could explain PRES associated with arterial hypertension, glomerulonephritis or autoimmune diseases such as thrombotic thrombocytopenic purpura, systemic lupus erythematosus, polyarteritis nodosa, cryoglobulinaemia, and among others systemic autoimmune diseases, pre-eclampsia/eclampsia or sepsis. Additionally, PRES has also been associated with cytotoxic drugs such as chemotherapies or immunosuppressants, and transplant recipients. Furthermore, the epidemiology of PRES is not well established and probably underestimated. The incidence of PRES among patients with end-stage renal disease and systemic lupus erythematosus accounts for less than 1% each, whereas in pregnant patients with pre-eclampsia or eclampsia, a high prevalence of PRES was found, with 90 and 20% respectively. Besides, the highest incidence appears to be among middle-aged adults and more prevalent in women (72% of admissions with a diagnosis of PRES in the USA)[1,3].

Magnesium is the second most abundant intracellular ion after potassium and the fourth most abundant ion in plasma. Its serum levels are tightly regulated due to its critical role in cellular homeostasis, vascular tone maintenance and endothelial integrity. While the relationship between hypomagnesaemia and PRES is not yet fully understood, it has been documented in isolated cases, particularly in the context of malabsorption syndromes such as short bowel syndrome[6], or in cases of malnutrition such as heavy drinkers, as recently reported[7].

In this case, the diagnosis of PRES was established based on well-defined clinical and neuroimaging criteria, with imaging abnormalities demonstrating reversibility

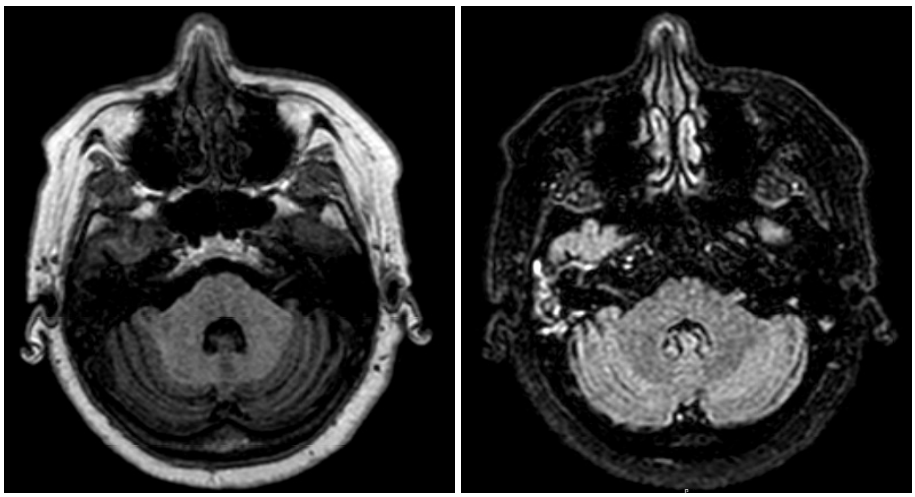


Figure 3. Brain MRI two months after discharge, showing complete neuroimage recovery.

following appropriate therapeutic intervention. Severe hypomagnesaemia, attributed to multifactorial origins, was identified as the main precipitating factor after the exclusion of alternative aetiologies.

CONCLUSIONS

PRES represents a diagnostic challenge by mimicking severe neurovascular pathologies. Without prompt and accurate diagnosis, it may lead to an increased risk of morbidity and mortality. Besides, hypomagnesaemia represents an uncommon but potentially reversible cause of PRES and should be taken into account for differential diagnosis.

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