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PPI Induced Hypomagnesemic Hypoparathyroidism Leading to Posterior Reversible Encephalopathy Syndrome Anam Malik MD, Shannon Davis DO, Yehia Mishriki MD Department of Internal Medicine, Lehigh Valley Health Network, Allentown, Pennsylvania

INTRODUCTION

- Hypomagnesemia is a well-known complication of proton pump inhibitor (PPI) use.^{1,2}
- Magnesium plays an important role in vascular endothelial regulation. Deficiency can lead to cerebral edema and posterior reversible encephalopathy (PRES).³

CASE REPORT

- A 42 year old male presented with anorexia, dyspepsia and nausea.
- His medication list was notable for chronic PPI use. Physical exam at that time was normal.
- Initial laboratory findings were normal with the exception of a calcium of 7.5mg/dl.
- He subsequently presented with a severe headache and dizziness. Laboratory studies revealed a calcium of 6.5 mg/dl, magnesium 0.4mg/dl and phosphorus of 3.8 mg/dl. A repeat calcium was 5.5 mg/dl. Urinary calcium was <5.0 mg/dl. Fractional urinary excretion of magnesium was 0.2%. A PTH level was normal.
- MRI of the brain showed bilateral cerebellar vasogenic edema. The differential diagnosis included posterior reversible encephalopathy syndrome (PRES) versus acute cerebellitis. EBV, Bartonella and Lyme serologies were negative. ANA testing was negative.
- The PPI was discontinued. The magnesium level normalized with replacement and the calcium level rapidly normalized. His symptoms quickly resolved.
- A repeat MRI showed significant improvement in the cerebellar edema.





DISCUSSION

- Hypomagnesemia can present with a variety of symptoms including seizures, cardiac arrhythmias, and neurological deficits.³
- Magnesium is known to have a role in the regulation of vascular tone and reactivity.¹
- Hypomagnesemia can cause endothelial dysfunction with capillary leakage, blood brain barrier disruption and axonal swelling leading to cerebral edema.
- Posterior reversible encephalopathy syndrome is due to disordered cerebral autoregulation and endothelial dysfunction.
- Impaired active absorption of magnesium by intestinal epithelial cells is caused by PPI-induced inhibition of transient receptor potential melastatin TRPM6 and TRPM7 channels.⁴
- Hypomagnesemia also impairs release of PTH and decreases peripheral tissue sensitivity to PTH leading to a functional secondary hypoparathyroidism.⁵
- Our patient improved with magnesium repletion which is compatible with secondary hypoparathyroidism and reversible cerebellar findings due to PPI induced hypomagnesemia.

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