Severe Hypocalcaemia and Hypomagnesaemia Secondary to Omeprazole

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Introduction

Proton pump inhibitors (PPIs) are commonly prescribed drugs. We report a case of severe hypocalcaemia secondary to omeprazole induced hypomagnesaemia.

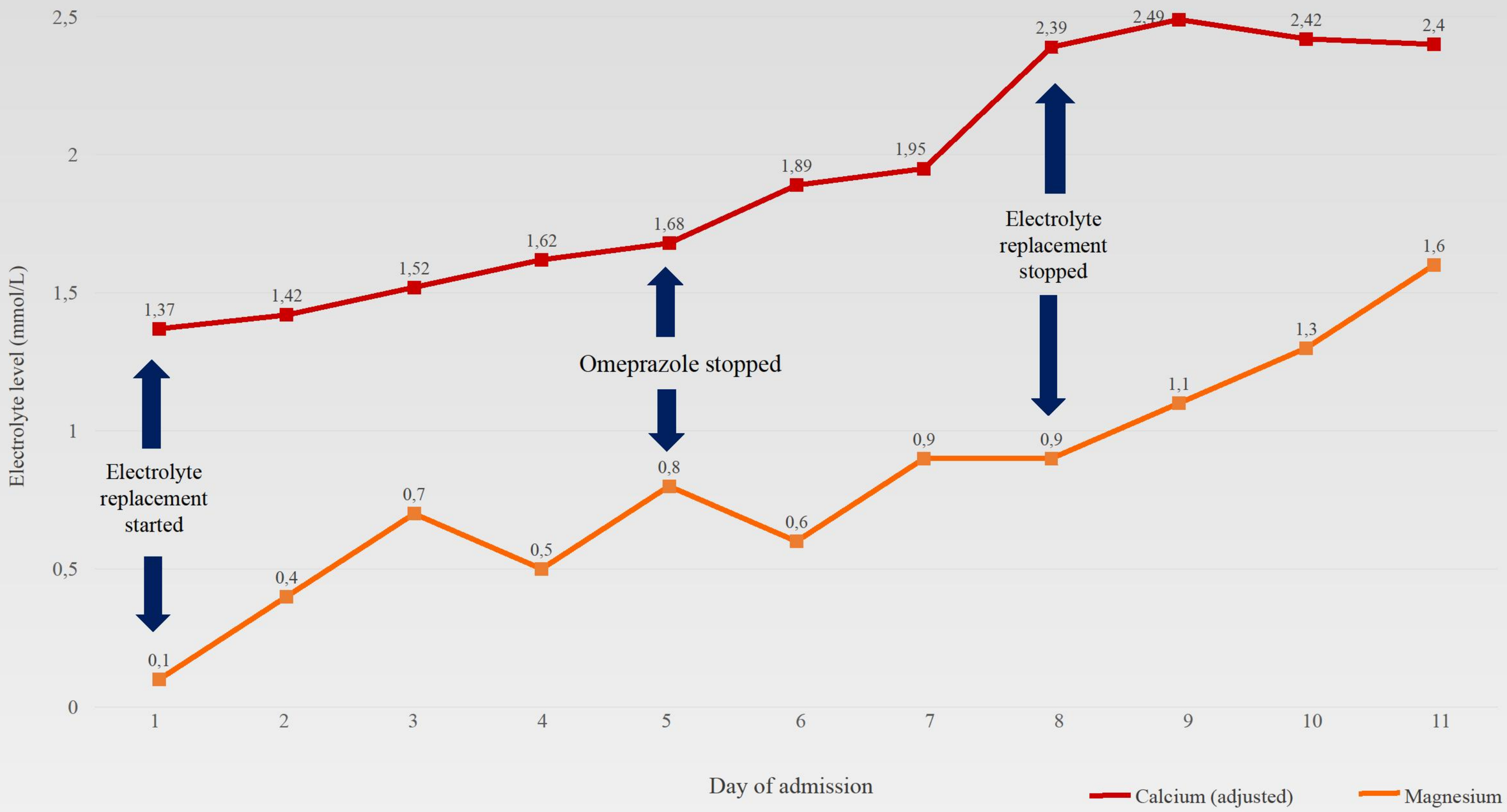
Case Report

An 85 year old Chinese man with stage 4 chronic kidney disease was admitted to our hospital in Jun 2014 with vomiting, loss of appetite and generalized weakness. On admission, he was severely hypocalcaemic (adjusted calcium 1.37 mmol/L [RI: 2.15-2.58]) and hypomagnesaemic (Mg 0.1 mmol/L [RI: 0.7-1.0]). Serum phosphate and 25-OH Vitamin D levels were normal. Intact PTH done after normalisation of serum magnesium showed secondary hyperparathyroidism (intact PTH 61.0 pmol/L [RI: 0.8-6.8]).

An inpatient Endocrinology referral was made for persistent hypocalcaemia despite aggressive intravenous replacement over 3-4 days. Review of his medical records revealed initiation of omeprazole in 2010 for peptic ulcer prophylaxis when he was prescribed short course prednisolone for a gout flare. Omeprazole was continued after prednisolone cessation. Severe hypomagnesaemia and hypocalcaemia was also noted during 2 previous admissions to another hospital in Nov 2013 and Jun 2014. Calcium levels were normal between hospitalisations but magnesium levels were not rechecked.

In view of the possibility of omeprazole induced hypomagnesaemia and hypocalcaemia, omeprazole was stopped and changed to famotidine. With electrolyte replacement and a short course of calcitriol, serum calcium normalised within 4 days and remained normal at follow-up with calcium carbonate 1.25 g bd (as a phosphate binder) and no magnesium supplementation.

Trend of Calcium and Magnesium levels



Conclusion

Severe hypomagnesaemia with hypocalcaemia is a rare side effect of PPI use[1]. Magnesium absorption is achieved by active saturable transcellular transient receptor potential melastatin 6 and -7 (TRPM6 and -7) channels and by non-saturable paracellular passive diffusion. TRPM6 and -7 protein channels are responsible for active transport of divalent cations including magnesium, calcium, zinc, cobalt, manganese and nickel. The highest affinity is for magnesium cations. Luminal acidity along the gastrointestinal tract is important in magnesium absorption as TRPM transporters and paracellular pores are pH sensitive[4]. PPI use may alter intestinal absorption of magnesium, resulting in hypomagnesaemia, which then in turn impairs PTH secretion and action, causing hypocalcaemia[2]. Magnesium acts as a neuromuscular stabilizer by interfering with acetyl choline release in the extracellular space and by inhibiting release of calcium by myocyte sarcoplasmic reticulum. Correction of magnesium levels by stopping the PPI and aggressive magnesium replacement is hence essential before correction of the hypocalcaemia can be achieved[3]. It is therefore important to have a high index of suspicion for this potentially life threatening side effect in patients on omeprazole presenting with hypocalcaemia.

References

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