Severe Hypophosphataemia Induced by Intravenous Iron

Role of the Pharmacist in Searching For Alternate Treatments

Jacky Hanh1, Benjamin Kwan1,2, James Lawrence Cowlishaw1

1 Concord Repatriation General Hospital, Sydney, Australia, 2 University of Sydney, Sydney, Australia

Background

Hypophosphataemia is an uncommon adverse effect associated with intravenous iron.[1] This case report uses the concept of calcitriol to treat a patient with severe hypophosphataemia refractory to intravenous phosphate supplement.

Clinical Features

A 72-year-old female with a history of scleroderma and oesophageal strictures requiring frequent oesophageal dilatation presented to hospital with severe vomiting. She also presented with generalised weakness, fatigue, body aches and paraesthesia in both hands. Her medical history included iron deficiency anaemia managed by frequent iron infusions with the last infusion administered three weeks ago.

Laboratory results revealed severe hypophosphataemia (serum phosphate 0.18 mmol/L). Other results were consistent with intravenous iron induced hypophosphataemia mediated by elevated fibroblast growth factor 23 (FGF23) including:

- 1,25-dihydroxyvitamin D deficiency
- Phosphaturia
- Hypocalcaemia

Elevated parathyroid hormone (PTH) measured 23.2 pmol/L (Table 1). Intravenous phosphate replacement (30 to 40 mmol/day) was insufficient in correcting serum levels.

Case Progress

The pharmacist performed a literature search to identify alternatives and recommended the use of calcitriol to the treating team as three case reports described the use of calcitriol with good effect.

Treatment with calcitriol 0.25 micrograms daily for three days, then twice daily thereafter resulted in a rapid increase in serum phosphate to 0.71 mmol/L within five days. Phosphate levels were maintained without the need for further intravenous phosphate replacement.

Parathyroid hormone levels also normalised by day five of calcitriol treatment. The patient displayed pronounced improvements in symptoms and was discharged 12 days after calcitriol started.

Mechanism of Hypophosphataemia

Calcitriol increases renal reabsorption of PO4, while PTH decreases it. Hypophosphataemia is induced by an increase in FGF23 which is mediated by a rise in intravenous iron. This case highlights the role of the pharmacist to proactively identify alternate treatments.

Conclusion

Severe hypophosphataemia induced by intravenous iron is an uncommon complication and can be refractory to intravenous phosphate replacement. Calcitriol may be effective in correcting hypophosphataemia, particularly in cases refractory to intravenous phosphate replacement. This case highlights the role of the pharmacist to proactively identify alternate treatments.

References


