

A Novel Case of Hypomagnesaemia Secondary to Fludrocortisone

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Case History

27 year old female with known autoimmune polyglandular syndrome, type 1, admitted with carpopedal spasm on a background of one week abdominal pain. Patient denied vomiting or diarrhoea. Investigations showed no acid-base disturbance or evidence of inflammation or infection.

Table 1: Admission medication list for case patient

Drug	Dose
Fludrocortisone	200 mcg OM 100 mcg ON (added 4 m earlier)

Alfacalcidol 4 mcg OD

Levothyroxine 100 mcg OD

Table 2: Serum chemistry results throughout admission

Blood results	Admission	Discharge (Day 3)
Magnesium	0.53 mmol/L	0.72 mmol/L
Potassium	3.1 mmol/L	3.2 mmol/L
Calcium	1.94 mmol/L	2.43 mmol/L
Ionized Calcium	0.88 mmol/L	N/A

References

1. Romani AM. Cellular Magnesium homeostasis. Arch Biochem Biophys 2011;512:1

2. https://www.medicines.org.uk/emc/medicine/30358

3. Quamme GA. Renal Magnesium handling: New insights in understanding an old problem. Kidney Int 1997;52:1180

Treatment

Initial treatment was intravenous electrolyte replacement followed by discontinuation of evening fludrocortisone. The mainstay of electrolyte replacement was Magnesium followed by Potassium and Calcium.

Proposed Mechanism

Magnesium is a mainly intracellular orphan element involved in over 300 chemical reactions in human physiology¹.

The Summary of Product Characteristics for Fludrocortisone does not mention hypomagnesaemia as a potential side effect² and an Ovid Medline literature search reveals no reports of this occurring. However, we propose that Fludrocortisone effects the renal handling of magnesium. Approximately 80% of plasma magnesium passes to the nephron via the glomerulus, the majority being absorbed via the paracellular

route at the thick ascending limb of the loop of Henlé, this is dependent on an electrical gradient across the membrane. The remaining 5-10% of magnesium is absorbed in the distal convoluted tubule, DCT, via the transcellular route and is dependent on potassium secretion into the distal tubule to create a voltage gradient³. Fludrocortisone is known to produce hypokalaemia through its effects in the DCT and therein may lie the mechanism for its hypomagnesaemic effects.

Learning Points

1. Electrolyte studies including Magnesium and Calcium should be checked regularly in patients taking Fludrocortisone, especially after dose changes
2. Further evaluation of the exact mechanism of action would be key to better understanding why this observation may occur

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