

# Hypomagnesaemia and the burn patient

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## Introduction

Burn injuries result in a number of physiological derangements. In the initial phase post burn injury, it is common to see hyponatraemia and hyperkalaemia which are then often followed by hypocalcaemia, hypophosphataemia and hypomagnesaemia.<sup>1</sup> If uncorrected, electrolyte derangements result in a range of clinical manifestations including cardiac, musculoskeletal and neurological problems. By the rehabilitation phase, electrolyte derangements seem to have resolved with minimal surface area wounds still unhealed. We have noticed however that magnesium may continue to drop in some patients in our unit and we wished to make other clinicians managing burns aware of our experience for the benefit of their patients.

Various studies have shown deficit of magnesium in burns patients: Toppo et al found that on day 10 post burn injury, 48% of cases had hypomagnesaemia and 32% had magnesium deficiency syndrome<sup>2</sup>; in another older study 8 out of 20 patients measured were magnesium deficient.<sup>3</sup> Our unit routinely measures and aggressively replaces magnesium in the acute phase of injury. We have also observed persistent hypomagnesaemia in the later stages of recovery. Recognising hypomagnesaemia through correlation with symptoms and replacing such deficits is crucial in the management of burn-injured patients. We would like to highlight the causes and effects of hypomagnesaemia as a means to encourage clinicians looking after these patients to look for and correct the abnormality.

## Role of magnesium

Magnesium is essential for nerve conduction, the active transport of potassium and calcium and therefore the regulation of blood pressure and heart rate. It is also involved in muscle contraction and relaxation as well as glucose control, oxidative phosphorylation, glycolysis and protein synthesis.<sup>4</sup> Deficiency can result in a wide range of symptoms including poor memory, delirium, psychiatric disturbances, muscle tremors and tetany, and cardiac problems such as dysrhythmias.

The majority of magnesium is found in bone and muscle with < 1% found in serum, making serum levels a poor reflection of total body magnesium. It is however still routinely used to detect deficiency in patients, with a level of < 0.7 mmol/l being considered low.<sup>4</sup> Serum magnesium is regulated by intestinal absorption and renal excretion. Low magnesium may be a result of excessive loss, failure of absorption or deficient intake.

## Magnesium and burns

Excessive loss may occur via the burn wound exudate or through urinary losses. This usually correlates with burn severity.<sup>5</sup> Loss through the kidneys can be caused by glomerular necrosis as a result of hypovolaemia and shock during the acute stages of burn injury. Endocrine abnormalities have also been found to cause an increase in renal excretion of magnesium; these include high cortisol and high aldosterone levels. A 24-hour urinary magnesium value of > 6.5 mmol/l is considered high.<sup>6</sup> This can be a useful investigation when determining the cause of hypomagnesaemia. The other two endocrine causes which are linked to magnesium abnormalities and are known to be altered in burns patients include thyroid and parathyroid hormones. In a review, Klein et al suggested that magnesium deficiency may be due to the hypermetabolic response of burns resulting in increased requirements of magnesium and increased uptake into the cell.<sup>7</sup> Other causes of low magnesium levels include medications, for example aminoglycosides, proton pump inhibitors and diuretics.<sup>4</sup> Serum magnesium is also known to drop in refeeding syndrome. This is a possibility which should be considered in burn patients who have been starved for a prolonged period or who are malnourished. Reduced magnesium intake and malabsorption could also cause deficiency as periods of starvation and feed intolerance are common in this group of patients. Lastly, a portion of serum magnesium is bound to albumin, which is commonly low in burn patients, and this may then reflect as low magnesium.<sup>4</sup>

We understand the reasons for low magnesium in the acute phase of burn injury but have seen deficiency even in the later phases in some patients, when many of the reasons described above do not apply. In these cases patients have only small wounds, are not on the medication usually implicated and have been receiving and tolerating their requirements for an extended period. They are no longer on IV fluids and albumin levels are on the upward trend.

Cuthbertson described the process of ebb, flow and recovery which occurs post injury where the flow stage is associated with a catabolic stage and, once in recovery, an anabolic state.<sup>8</sup> In an anabolic state during the recovery phase of burns it is presumed that magnesium uptake would increase for protein synthesis and could explain the low serum levels. This theory is illustrated by repeated low serum magnesium in the presence of magnesium replacements and an upward trend of albumin. Graph 1 is an example of the trend of albumin and magnesium in the recovery phase of a patient with 47% total body surface area burns in our unit.

### Management of hypomagnesaemia

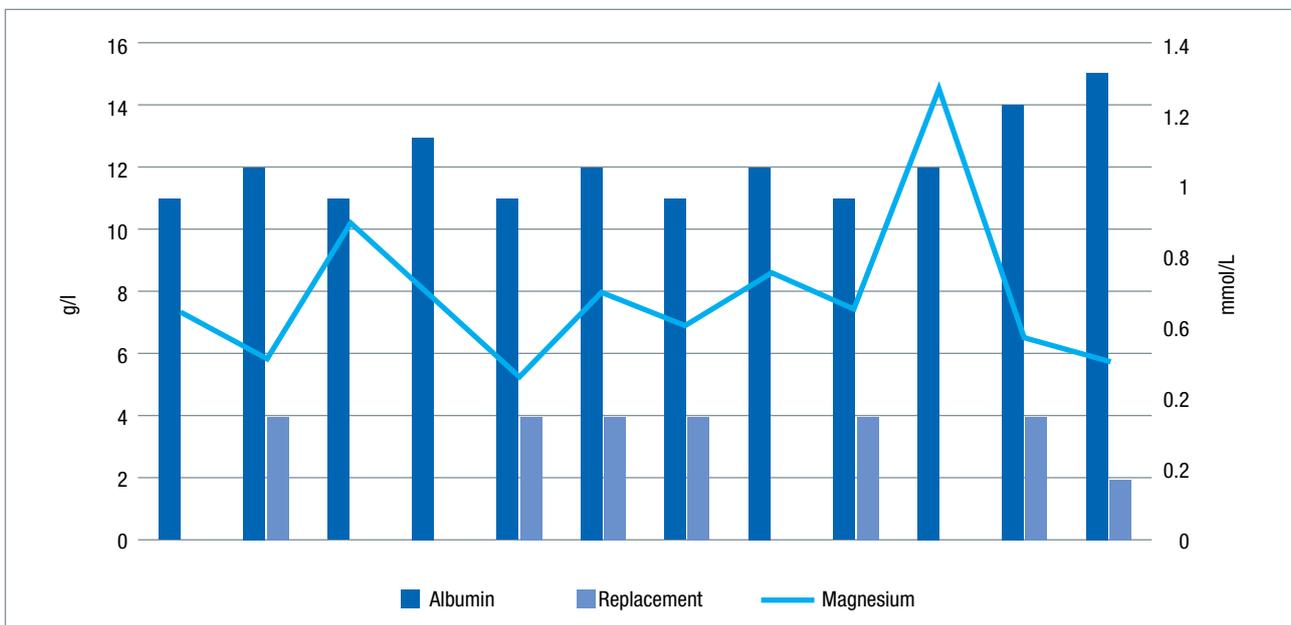
Magnesium repletion depends on the clinical scenario. In our service, we replace magnesium intravenously at a dose of 50 milligrams per kilogram in children and four grams in adults daily, until corrected,<sup>9,10</sup> in both the acute and late phases. In patients who have had magnesium deficiencies we check the levels three times a week as opposed to those patients with no electrolyte abnormalities, where bloods are done weekly.

### Conclusion and recommendations

Magnesium deficiency is multifactorial in the acute phase of burn injury but can persist into the recovery period of treatment. Magnesium is an essential electrolyte in many physiological pathways. We wish to highlight the importance of checking serum magnesium levels regularly and that replacement may be necessary throughout the admission period.

### References

- Ramos CG. Management of fluid and electrolyte disturbances in the burn patient. *Annals of Burns and Fire Disasters*. 2000;13(4):201-5.
- Toppo D, Kamal D, Pratap D, Kant D. Observations of serum magnesium levels in major burns. *IOSR Journal of Dental and Medical Sciences*. 2017;16(05):22-26.
- Broughton A, Anderson I, Bowden C. Magnesium-deficiency syndrome in burns. *The Lancet*. 1968;292(7579):1156-1158.
- Jahnen-Dechent W, Ketteler M. Magnesium basics. *Clinical Kidney Journal*. 2012;5(Suppl 1):i3-i14.
- Berger M, Rothen C, Cavadini C, Chioloro R. Exudative mineral losses after serious burns: a clue to the alterations of magnesium and phosphate metabolism. *American Journal of Clinical Nutrition*. 1997;65:1473-1481.
- Milan A. [Internet]. [cited 20 August 2017]. Available from <http://pathlabs.rlbuht.nhs.uk/magnesium.pdf>
- Klein G, Herndon D. Magnesium deficit in major burns: role in hypoparathyroidism and end-organ parathyroid hormone resistance. *Magnesium Research*. 1998;11(2):103-109.
- Cuthbertson D. Post-shock metabolic response. *The Lancet*. 1942;239(6189):433-437.
- Magnesium sulphate. [cited 29 November 2017]. Available from <http://bnf.nice.org.uk>
- Clinical fluid and electrolyte management. [cited 29 November 2017]. Available from <http://seattlechildrens.org>



Graph 1. Albumin (g/dL) and magnesium (mmol/L) trend in a single patient in recovery phase