Information for Clinicians

Clinical Biochemistry Department

Hypomagnesaemia – a guide for GPs

Amendment History

<table>
<thead>
<tr>
<th>Issue</th>
<th>Status</th>
<th>Date</th>
<th>Reason for Change</th>
<th>Authorised</th>
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<tbody>
<tr>
<td>1.0</td>
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<td>Oct 2021</td>
<td>First issue</td>
<td>M. O’Doherty</td>
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<tr>
<td>2.0</td>
<td>Approved</td>
<td>June 2024</td>
<td>Scheduled review</td>
<td>M. O’Doherty</td>
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Overview
Magnesium is abundant in foodstuffs such as green leafy vegetables as Mg is a component of chlorophyll. Most people will take in more (typically 10-12 mmol/24h) than the minimum daily requirement of approximately 8 mmol/24h.

Normal range and concentrations associated with mild, moderate and severe deficiency:

Mild deficiency: 0.5-0.7 mmol/L – only require oral replacement if symptomatic

Moderate deficiency: 0.4-0.5 mmol/L – advise oral replacement if asymptomatic, IV replacement if symptomatic

Severe deficiency: <0.4 mmol/L – usually requires admission for IV replacement

All results ≤0.4 mmol/L will be phoned by the laboratory to the requesting location or out of hours (OOH) service, consider urgent admission in these cases (especially if acute onset or symptomatic).

GP Acute Medical Referrals and MAU Consultant Advice Line: 07818 013823

Symptoms of low magnesium (typically occur when Mg <0.5 mmol/L) include:

- Nausea, vomiting
- Lethargy, muscle weakness, drowsiness
- Tetany, tremor, twitching, agitation
- Vertigo
- Confusion
- Cardiac arrhythmias
- Seizures
Symptomatic hypomagnesaemia is often associated with:
- Hypocalcaemia (Mg is required for PTH secretion)
- Hypokalaemia (Mg is required for normal functioning of the Na⁺/K⁺ transmembrane ATPase).

**Causes of low magnesium**

GI-related:
- Diarrhoea and vomiting
- Malabsorption Medications (e.g. PPIs, laxatives)
- Gastrointestinal fistulae
- Intestinal resection/Short bowel syndrome
- Malnutrition/dietary deficiency (uncommon)

Renal losses:
- Medications (e.g. thiazide or loop diuretics, amphotericin, aminoglycosides, immunosuppressants, cisplatin, cyclosporin, tacrolimus)
- Diabetes Mellitus/Glycosuria (osmotic diuresis)
- Alcoholism
- Hypercalcaemic states
- Hyperaldosteronism
- Renal tubular disorders
- Inherited disorders e.g. Bartter’s, Gitelman’s syndrome
- Post renal transplant
- Dialysis

Redistribution:
- Refeeding syndrome

NB: Severe hypoalbuminaemia may result in low serum magnesium but this is physiologically appropriate due to a proportion of circulating magnesium being bound to albumin.

**Investigations**

In most patients with hypomagnesaemia the cause will be obvious from the history; the underlying cause should be treated.

If hypomagnesemia is persistent, and the cause is unclear, 24 hour urine samples for magnesium may be used to assess whether magnesium is being lost renally.

The normal renal response to hypomagnesaemia is to lower magnesium excretion to very low levels.

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<th>Urine loss/24 hours</th>
<th>Interpretation in presence of hypomagnesaemia</th>
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<tr>
<td>&gt;0.5 mmol (&gt;12 mg)</td>
<td>Renal wasting</td>
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A fractional excretion of magnesium (FEMg) >2% in a subject with normal renal function indicates renal magnesium wasting.
To discuss specific cases the Duty Clinical Biochemist may be contacted on 01225 824050 (available 9 am-5 pm Mon-Fri).

NB: Ensure that potassium and calcium have been checked following a finding of hypomagnesemia due to the risk of hypokalaemia or hypocalcaemia developing.

**Management**

- If symptomatic and especially if the magnesium is <0.4 mmol/L recommend urgent admission.
- For asymptomatic or mild hypomagnesaemia review the patient for the underlying cause. Most commonly this will be due to recent losses i.e. diarrhoea or medications.
- If appropriate, stop medications which may cause hypomagnesaemia.
- Treatment is not always required if mild hypomagnesaemia and some patients with chronically low levels may tolerate magnesium that rests just below the reference range (for example with short bowel syndrome or Gitelman’s syndrome).
- See below for replacement regime.
- Potassium (and magnesium)-sparing diuretics (e.g. amiloride) should be considered in patients with diuretic-induced hypomagnesemia (when diuretic therapy cannot be discontinued) or in cases of chronic renal magnesium wasting.

**Replacement**

Magnesium may be given orally in a dose of **up to 24 mmol Mg^{2+}** daily in divided doses.

Avoid or significantly reduced (50%) doses of Mg salts in patients with reduced renal function (eGFR <30 mL/minute/1.73^2) and monitor closely. A rare compilation of supplementation is hypermagnesaemia (see below).

Treatment is often poorly tolerated being limited by diarrhoea so advise to take with or after food.

<table>
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<th>Serum Mg(mmol/L)</th>
<th>Replacement options</th>
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<tr>
<td>0.5-0.7 <strong>with</strong> symptoms</td>
<td>Magnesium aspartate (Magnaspartate®) 1 sachet (10 mmol) BD (standard option)</td>
</tr>
<tr>
<td><strong>OR</strong></td>
<td>OR</td>
</tr>
<tr>
<td>0.4-0.5 <strong>without</strong> symptoms</td>
<td>Magnesium glycerophosphate (Neomag®) 1-2 x 4 mmol chewable tablets TDS</td>
</tr>
<tr>
<td>0.4-0.5 <strong>with</strong> symptoms</td>
<td></td>
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<tr>
<th>OR</th>
<th>Admission for IV Magnesium sulphate</th>
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<tr>
<td>&lt;0.4 with/without symptoms</td>
<td></td>
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Monitoring is dependent on the clinical scenario and symptoms but in general repeat serum magnesium initially after 5-7 days of replacement. Serum magnesium levels rise quickly with treatment but intracellular magnesium takes longer to replenish – continuation of replacement for a further 2 days after normalisation of levels should be considered in patients with normal renal function.

Please see the BaNES, Swindon and Wiltshire Joint Formulary for details of locally available supplements to treat magnesium deficiency: [http://www.bswformulary.nhs.uk/about.asp](http://www.bswformulary.nhs.uk/about.asp)

**Hypermagnesaemia**

The kidneys normally excrete excess magnesium very efficiently, so hypermagnesaemia is largely restricted to patients with acute or chronic renal failure. However, raised magnesium levels may also be seen in patients when magnesium replacement is given or in tumour lysis syndrome.

Hypermagnesaemia in the order of 1.5-2.5 mmol/L may be associated with hypotension but is commonly asymptomatic. In the range 2.0-5.0 mmol/L, areflexia may be present and electrocardiographic changes can occur. At higher concentrations, respiratory arrest and paralysis ensue.

In the very rare likelihood of encountering a patient with hypermagnesaemia consider discussion with biochemistry or the on-call medical team.

**References**


