HYPOMAGNESAEMIA
Guidance for the assessment and management in primary care

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General Principles

1. PRESENCE AND SEVERITY:

- URGENT secondary care referral is required in severe symptomatic hypomagnesaemia, for consideration of intravenous magnesium replacement.

2. IDENTIFY CAUSE:

- CAUSATIVE MEDICATION should be stopped if possible.

3. TREATMENT:

- Identify patients in need of urgent secondary care referral.
- Magnesium replacement ***may*** not be required if underlying cause is removed.
- Replacement must take into account renal function (reduce dose if eGFR <30mL/min/1.73m^2)
- Serum magnesium concentration (and any other relevant electrolytes) should be monitored regularly to assess the response to treatment.
- Intravenous therapy may also be needed in patients with impaired gastrointestinal absorption, severe diarrhoea or those unable to tolerate oral supplements. Refer to a secondary care specialty appropriate to the underlying diagnosis.

DOCUMENT:

- The presence of hypomagnesaemia (blood test result), the underlying cause/diagnosis and the immediate and longer term management plan.

MAGNESIUM TOXICITY:

- If treatment-related magnesium toxicity (i.e. hypermagnesaemia) is suspected, treatment should be discontinued. The elderly and patients with renal insufficiency are at increased risk. Clinical symptoms and signs of toxicity include:
  - Hypotension, bradycardia, respiratory depression, depressed mental state, nausea and vomiting
  - ECG abnormalities (bradycardia, prolonged PR, QRS, QT, complete heart block, asystole)

Part 1. PRESENCE AND SEVERITY

Normal serum magnesium is 0.7 – 1.0 mmol/L

The *severity* of hypomagnesaemia should be defined by the **serum magnesium concentration** AND the **presence/absence of symptoms** AND/OR **ECG changes**.

1. **Serum magnesium concentration**: Mild 0.5-0.69 mmol/L
   Severe <0.5 mmol/L

2. **ECG changes**:

Prolonged PR interval, widened QRS complex, prolonged QTc, flattened T wave, atrial / ventricular dysrhythmias (especially if on digoxin), polymorphic ventricular tachycardia (torsade de pointes).

The presence of ECG changes indicates clinically significant hypomagnesaemia and the potential for serious (life threatening) dysrhythmias.

3. **Signs and symptoms**:

Musculoskeletal	Muscle twitching, tremor, tetany, cramps, seizures
CNS	Apathy, depression, hallucinations, agitation, confusion
Cardiovascular	Tachycardia, hypertension, dysthymias, digoxin toxicity
Biochemical*	Hypokalaemia, hypophosphataemia, hypocalcaemia, hyponatraemia

Hypomagnesaemia rarely occurs in isolation and other electrolyte levels **(potassium, phosphate, calcium, sodium) should also be measured. If hypokalaemia does not respond to supplementation, always check for a decreased magnesium level.*

Part 2. IDENTIFYING THE CAUSE

The successful management of hypomagnesaemia is dependent on the correct identification of the underlying cause (see table below).

Medications are a common (probably the commonest) cause. **Proton pump inhibitors** and **diuretics** are frequently implicated.

Take a history and assess the patient. This will usually reveal the cause. If the cause is not clear arrange a 24 hour urine collection for urine magnesium (In renal magnesium wasting the 24 hour urine magnesium will be >1mmol/24hours; in gastrointestinal magnesium wasting the 24 hour urine magnesium will be <1mmol/24hours).

Causes of hypomagnesaemia:

Medications	**Renal causes**	**GI causes (Reduced absorption)**
Proton Pump Inhibitors*	***Alcohol misuse****	***Chronic Diarrhoea**** / Laxative abuse
Diuretics* (thiazides or loops)	Uncontrolled diabetes mellitus	*Alcohol misuse**
Aminoglycosides	Volume expansion	Malabsorption (e.g. Coeliac Disease)
Amphotericin	Hypercalcaemia (PHP)	Vomiting
Ciclosporin, Tacrolimus	Acquired tubular dysfunction	Refeeding syndrome
Theophylline	Recovery from acute tubular necrosis	Bowel Fistula
Salbutamol	Post-obstructive diuresis	Short Bowel Syndrome
Cytotoxics (Cisplatin)	Post-renal transplant	Acute pancreatitis
	Genetic disorders (e.g. Bartter/Gitelman syndrome)	Reduced intake (dietary deficiency –rare)
	24 hour urinary Magnesium	
	>1mmol/24h	<1mmol/24h

**** Common cause***

Part 3. TREATMENT

Step 1. Identify whether or not URGENT treatment is needed:

Patients with severe hypomagnesaemia, as defined by the serum magnesium AND the presence of symptoms and/or ECG changes are likely to require URGENT admission to hospital for intravenous magnesium.

Patients with moderate or mild hypomagnesaemia (defined by serum magnesium AND the absence of ECG changes / significant symptoms) can be safely managed in primary care.

Step 2. Management of moderate or mild hypomagnesaemia in primary care:

If possible correct the underlying cause (e.g. stop causative medications, support alcohol cessation, treat diarrhoea, etc).

Consider oral magnesium supplements to restore the serum magnesium to the normal range (see box). These may not be required at all or may not need to be continued if the underlying cause has been corrected.

If oral therapy is not tolerated (the commonest side effect is diarrhoea, which can further exacerbate hypomagnesaemia) OR serum magnesium is not responding to treatment OR hypomagnesaemia recurs despite oral treatment, refer to the relevant specialty according to the suspected underlying cause. This is particularly important if long term intravenous magnesium is likely to be required.

Oral magnesium supplements

1. **Normal kidney function**: Magnesium aspartate (Magnaspartate) is the preferred oral therapy. One sachet contains 10 mmol Mg^{2+}. This can be given as 1 sachet once daily or 1 sachet twice daily depending on severity of deficiency. **Monitor serum [Mg] at appropriate intervals to determine response and need for on-going therapy.**
2. **Chronic kidney disease:** If eGFR is < 30 mL/min/1.73 m^2 there is a risk of severe hypermagnesemia when prescribing oral Mg^{2+} supplements. Only prescribe supplements if essential and give the lowest dose possible (reduce usual dose by at least 50%). **Monitor serum magnesium level at appropriate intervals to determine response and the need for on-going therapy**.