MIL

Oxford Radcliffe Hospitals NHS
NHS Trust

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This Medicines Information Leaflet is produced locally to encourage prescribing that is cost effective to the NHS. Information will be given on quality improvement issues and the costs to hospital and community.

Guidelines for the Management of Hypophosphataemia in Adults

Within ORH Trust hypophosphataemia is defined as a plasma phosphate level of less than 0.7mmol/L. This MIL summarises the preparations and methods of phosphate supplementation – both oral and intravenous – recommended in this trust.

Definition

Mild hypophosphataemia:	0.61-0.69 mmol/L
Moderate hypophosphataemia:	0.41-0.60 mmol/L
Severe hypophosphataemia:	less than 0.40 mmol/L

Phosphorus is one of the most abundant elements in the human body, approximately 85% of which is contained in the skeleton. Most phosphorus in the body is complexed with oxygen as phosphate (PO_4).

In soft tissues phosphate is predominantly found in the intracellular compartment, it is a key component of cell membrane phospholipids and nucleic acids and is involved in aerobic and anaerobic energy metabolism (NAPD). Insulin drives phosphate intracellularly, therefore transient hypophosphataemia is associated with raised insulin levels either endogenous or exogenous.

Phosphate is well absorbed from the diet, and excreted renally. Phosphate depletion can occur in a number of diseases and the kidneys compensate by conserving phosphate to maintain a normal phosphate level. Phosphate accumulation also occurs in renal disease resulting in high plasma levels.

Causes of hypophosphataemia

Hypophosphataemia occurs in approximately 2% of hospitalised patients, however clinically important hypophosphataemia requiring treatment affects relatively few patients.

Causes of acute hypophosphataemia:

- Recovery phase of diabetic ketoacidosis
  Up to 90% of patients will experience moderate hypophosphataemia within 6-12 hours of starting therapy
  Supplementation in this setting is not recommended unless levels drop below 0.5mmol/L or remain low following stabilisation [1,2]
- Post partial hepatectomy and liver transplant
  Hypophosphataemia post hepatic surgery is common, and it considered to be due to transient excessive renal phosphate wasting [3], it is usually transient and rarely requires supplementation
- Acute alcoholism
- Re-feeding syndrome
  Feeding a patient after a significant period of starvation causes a rapid elevation of circulating insulin levels and can cause a transient hypophosphataemia [4]. Cautious re-introduction of nutrition will attenuate this effect.
- Severe burns

Causes of chronic hypophosphataemia:

- Hyperparathyroidism
- Cushings syndrome
- Hypothyroidism
- Chronic aluminium ingestion (usually in the form of antacids)
- Chronic diarrhoea or vomiting

Signs and symptoms:

Hypophosphataemia is usually asymptomatic, however symptoms can include:

- Weakness
- Pins and needles (paraesthesis)
- Respiratory failure
- Heart failure
- Dysarthria
- Confusion / Seizure / Coma
- Rhabdomyolysis
- Haemolytic anaemia

Treatment of Hypophosphataemia

Management of the underlying cause should be considered in all cases.

Oral supplementation should be considered first line in all patients that can tolerate oral therapy and who do not have a sodium restriction.

Supplementation should commence within 48 hours of a level being checked. Low levels beyond this timeframe should be re-checked before supplementation.

Isolated low levels should be re-checked, to establish validity and a trend, before supplementation is considered.

<table>
<tr><th>Serum Levels</th><th>Moderate
Asymptomatic
0.41-0.6 mmol/L</th><th>Moderate
Symptomatic
0.41-0.6 mmol/L</th><th>Severe
Less than
0.4mmol/L</th></tr>
<tr><td>Patient able to tolerate oral or enteral therapy</td><td>Phosphate Sandoz Effervescent tablets
Two tablets twice daily (16 mmol phosphate per tablet) NB: also provides 20mmol sodium and 3mmol potassium per tablet

Use i.v. therapy if patient has diarrhoea or high output stoma.</td><td colspan="2">Oral therapy not appropriate.
Use i.v. therapy as below.</td></tr>
<tr><td rowspan="2">Patient on intravenous therapy only</td><td>20mmol phosphate as sodium glycerophosphate in 0.9% sodium chloride or 5% glucose over 12 hours.

Dilution volume 50ml or less must be administered via central access.</td><td colspan="2">20mmol phosphate as sodium glycerophosphate in 0.9% sodium chloride or 5% glucose over 6 hours.

Dilution volume 50ml or less must be administered via central access.</td></tr>
<tr><td colspan="3">Check plasma calcium, if high seek specialist advice prior to supplementation.

See phosphate monograph for further information on dilution and administration.
Do not recheck phosphate for at least 6 hours post infusion (to allow for distribution).
Half dose in renal impairment or for patients less than 40kg</td></tr>
</table>

Patient Monitoring

Plasma levels should be checked more than 6 hours following infusion to allow for redistribution into the intracellular compartment.

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Bibliography

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4. Marvin.V., May.C.,Livingstone.C.,Davis.J. (2007) Incidence of hypophosphataemia in patients on parenteral nutrition. Hospital Pharmacist 14;166-169