POSTOPERATIVE MAGNESIUM DEFICIENCY

N. J. Paymaster

SUMMARY

Prolonged nasogastric suction and the administration of magnesium-free parenteral fluids produced magnesium deficiency in a patient in the postoperative period. The symptoms of magnesium deficiency are usually overshadowed by those of the primary disease and diagnosis usually rests on the demonstration of low blood concentrations of the ion. Magnesium replacement must be kept in mind when treating patients with intravenous fluids for prolonged periods.

It is common knowledge that the serum electrolyte concentrations of patients receiving prolonged intravenous fluid therapy should be monitored carefully. In particular, attention is usually directed at keeping the principal cations, sodium and potassium and the anions, chloride and bicarbonate, within normal limits. Although a few cases of hypomagnesaemia (Fletcher et al., 1960; Flink et al., 1954; Kellaway and Ewen, 1954; Opie, Hunt and Finlay, 1964) have been reported it is possible that many more remain undetected, especially when it is appreciated that the usual intravenous fluid therapy regimes do not include the administration of magnesium. The following is a case report of magnesium deficiency resulting from prolonged nasogastric suction in association with the administration of magnesium-free parenteral fluids.

CASE REPORT

A 57-year-old male was admitted to a medical ward in a coma, with widespread bruising over body and limbs. For many years he had imbibed large quantities of alcohol and he had suffered from severe jaundice in 1960. Examination revealed a dry tongue and marked gaseous distension of his abdomen. An x-ray of his chest was normal. Blood electrolyte concentrations were: sodium 132 m-equiv/litre, potassium 3.4 m-equiv/litre, bicarbonate 22.5 m-equiv/litre; the urea concentration was 62 mg/100 ml (table I). Anaesthesia was induced with thiopentone 250 mg followed by suxamethonium 65 mg. An endotracheal tube was inserted and the patient ventilated with 33% oxygen in nitrous oxide. Pancuronium was used to obtain muscle relaxation. A large tear in the caecum and a retroperitoneal haematoma were found and a right hemicolectomy, an ileo-transverse anastomosis and a colostomy were performed. Blood loss was estimated at 2000 ml and approximately 1800 ml of protein containing fluid was aspirated from the peritoneal cavity. Two litre of blood, 1200 ml of plasma and 500 ml of dextran 70 in dextrose were infused during surgery. After operation, intermittent positive pressure ventilation was continued for 24 hours and the acid base balance monitored. The nasogastric aspirate was copious for the first 6 days after surgery. Oral feeding was commenced on the 2nd day after surgery and intravenous fluid administration, 3-3.5 litre per day (blood, saline, dextrose and Hartmann's solution, potassium chloride) continued for 13 days. Amino acid and sorbitol administration was started on the 7th day after operation, when the patient's haemoglobin concentration was 12.9


**Table I. Serum biochemical estimations during period of hospitalization.**

<table>
<thead>
<tr>
<th></th>
<th>On admission</th>
<th>5th day</th>
<th>10th day (operation)</th>
<th>17th day</th>
<th>20th day</th>
<th>24th day</th>
<th>31st day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium (m-equiv/litre)</td>
<td>132</td>
<td>118</td>
<td>132</td>
<td>131</td>
<td>135</td>
<td>135</td>
<td>134</td>
</tr>
<tr>
<td>Potassium (m-equiv/litre)</td>
<td>3.4</td>
<td>3.2</td>
<td>3.2</td>
<td>3.3</td>
<td>3.1</td>
<td>4.0</td>
<td>3.9</td>
</tr>
<tr>
<td>Bicarbonate (m-equiv/litre)</td>
<td>22.5</td>
<td>22.5</td>
<td>26</td>
<td>30</td>
<td>27</td>
<td>26.5</td>
<td>25.5</td>
</tr>
<tr>
<td>Urea (mg/100 ml)</td>
<td>13</td>
<td>26</td>
<td>62</td>
<td>23</td>
<td>20</td>
<td>13</td>
<td>14</td>
</tr>
<tr>
<td>Non-protein nitrogen (mg/100 ml)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>3.1</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Calcium (mg/100 ml)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>3.1</td>
<td>8.2</td>
<td>—</td>
</tr>
<tr>
<td>Magnesium (m-equiv/litre)</td>
<td>—</td>
<td>—</td>
<td>1.08</td>
<td>1.25</td>
<td>1.5</td>
<td></td>
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</tr>
</tbody>
</table>

G/100 ml, haematocrit 38.9% and total serum proteins were 5.2 g/100 ml (albumin 2.2g/100 ml).

On the 9th postoperative day (19th day after admission to hospital) the patient complained of sleepiness and appeared to be confused. The following day he was very sleepy to the point of falling asleep while talking. He was slightly confused, had muscle weakness, tremor and purposeless involuntary movements; his deep tendon reflexes were normal and the plantar responses were equivocal. Arterial pressure, pulse and body temperature were normal. His urine output for the previous 24-hr period was 1600 ml (specific gravity 1020). Blood examination showed sodium 135, potassium 3.1, bicarbonate 27 m-equiv/litre; urea and non-protein nitrogen concentrations were 20 and 27.5 mg/100 ml respectively, total protein 6.3 g/100 ml, bilirubin 0.6 mg/100 ml, s.g.o.t. 155 i.u. and s.g.p.t. 125 i.u., normal liver function tests, inorganic phosphate 3.1 mg/100 ml, calcium 8.2 mg/100 ml and magnesium 1.08 m-equiv/litre (normal 1.5 to 2.00 m-equiv/litre). A diagnosis of magnesium deficiency was made and magnesium therapy, consisting of 35 ml of 20% magnesium sulphate (MgSO₄) was given intravenously on that day together with 3000 ml of 5% dextrose, 500 ml of 0.9% saline, potassium chloride 7 g and 30 ml of Parentrovite (Bencard Lab.). On the next day he was given a further 10 ml of 20% MgSO₄ and 6 ml of 50% MgSO₄, potassium chloride 10 g, 20 ml of Parentrovite, 5% dextrose, 0.9% saline and Intralipid (Paines and Byrne) by intravenous infusion. Toward the end of the day he had stopped twitching and was rational, brighter and not sleepy. On the following day he continued to improve and he was given 6 ml of 50% MgSO₄, potassium chloride 6 g, 2000 ml of saline 0.9%, 1000 ml of dextrose 5% and 500 ml of Intralipid. The patient had received 125 m-equiv of magnesium over a period of 72 hours. On the 24th day in hospital his intravenous infusion was discontinued. On the 31st day his serum magnesium was 1.5 m-equiv/litre; fourteen months later it was 1.8 m-equiv/litre.

**Discussion**

This patient was admitted in a state of moderate dehydration. His oral intake over the next 22 days ranged from nothing (for 7 days) to a maximum of 1526 ml in any one day, with a total of just over 15 litre. His oral intake consisted almost entirely of water, tea, milk, fruit juice (squash) and toward the end of the period, Hycal (Beecham Lab.) and Complan (Glaxo Lab.). With the exception of milk which has a magnesium content of 1 m-equiv/10 ml, the magnesium content of these items is insignificant. The average normal daily intake of magnesium is about 20 m-equiv (Scott, 1973).

The volume of nasogastric aspirate during this period was just under 6 litres (which theoretically would contain 6 m-equiv of magnesium) and colostomy drainage was about 5 litre. The total of intravenous fluids (Mg²⁺ free) administered during this period was approximately 67 litre. It is clear that magnesium deficiency resulted from prolonged nasogastric suction with administration of magnesium-free parenteral fluids. (Amino acids, Sorbitol and the other intravenous nutritional fluids used lack magnesium.) Alcoholism may have been a predisposing factor (Heaton et al., 1962; Wacker and Parisi, 1968a).

Marked sleepiness, muscle weakness, tremor and purposeless involuntary movements were the predominant features of magnesium deficiency in this patient. Vertigo, irritability, psychotic behaviour and tetany were absent. It is likely that he would have developed tetany if magnesium deficiency had not been detected and replacement therapy commenced. Wacker and Parisi (1968b) regard latent or overt tetany as the most distinctive symptom in the course of magnesium depletion. A normal serum calcium concentration with normal blood pH and
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promt reversal by administration of magnesium but not by calcium, characterize this tetany (generalized tonic-clonic as well as focal seizures). The concentration of serum Mg\(^{2+}\) in this patient (1.08 m-equiv/litre) at the time he developed symptoms compares well with the generally accepted critical level of 1 m-equiv/litre. It must be remembered, however, that tetany or other overt manifestations of magnesium deficiency do not develop in all patients with such low concentrations. The amount of magnesium bound to protein and the intracellular concentration are of obvious importance in determining when symptoms develop. Hypokalaemia and significant hypocalcaemia may co-exist with magnesium deficiency in patients with excessive loss of gastro-intestinal fluids. Serum potassium and calcium concentrations in this patient were marginally less than normal when he developed magnesium deficiency.

He was given average daily potassium requirements but no calcium during the period of magnesium therapy.

Magnesium deficiency should be suspected in all patients with conditions known to be associated with it. The diagnosis is seldom suggested by symptoms which are often of secondary importance to those of the primary underlying disease. Wacker and Parisi (1968a) have listed causes of symptomatic hypomagnesaemia. Some of the important ones are: malabsorption syndromes, bowel and biliary fistulae, alcoholism, hyperthyroidism, diabetic coma, diuretic therapy, renal disease and porphyria.

Hypomagnesaemia may safely be treated by parenteral magnesium therapy if the calculated extracellular deficit of magnesium is replaced in divided doses over a 48-hr period. In the presence of very low initial levels (0.8 m-equiv/litre) intravenous magnesium sulphate (10, 25 or 50%) 2 m-equiv/kg may be given over 4 hours. In the less severe cases 0.25 to 0.5 m-equiv/kg/day may be administered intravenously until the serum magnesium is normal (Thompson and Wootton, 1970).

ACKNOWLEDGEMENTS

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REFERENCES