Hypomagnesaemia-hypocalcaemia syndrome in the postpartum period

A case report

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Summary

A Black woman had amoebic dysentery for 5 days before delivery of her 7th child. The day after delivery at home she was admitted to hospital with puerperal sepsis, vaginal bleeding and dysentery. The patient received gentamicin from the 4th to the 10th day after admission and the diarrhoea continued until the 11th day, when she developed tetany. Investigation revealed hypocalcaemia, hypomagnesaemia and hypokalaemia; urinary magnesium excretion was at the upper limit of the reference range; infusion of parathyroid hormone resulted in a 12-fold increase in urinary cyclic adenosine monophosphate excretion. Magnesium replacement therapy was instituted and normal serum calcium, magnesium and potassium levels were re-established on this treatment alone. These findings are interpreted as being consistent with magnesium depletion by diarrhoea and gentamicin therapy, resulting in the induction of a functional hypoparathyroidism.

Hypoparathyroidism is extremely rare during pregnancy and the postpartum period. The circumstances under which it occurs and the mechanism involved are therefore of some general interest. This report deals with a patient who developed functional hypoparathyroidism secondary to hypomagnesaemia in the postpartum period.

Case report

A Black woman, 27 years old, was admitted to King Edward VIII Hospital, Durban, on the day after delivery of her 7th baby. She had had diarrhoea for 5 days before delivery and severe vaginal bleeding following the birth of her child. On examination she was pyrexial and dehydrated; her pulse rate was 120/min and her blood pressure 90/50 mmHg. There was generalized tenderness over the lower abdomen and a subinvoluting uterus was palpable. Infected products of conception were found in the vagina. Laboratory investigation at this time revealed: haemoglobin concentration 6,5 g/dl; platelet count 18 000/μl; white cell count 13 000/μl; and blood urea level 17 mmol/l. The patient was resuscitated by red blood cell transfusion and fluid and electrolyte therapy and immediate treatment with chloramphenicol and metronidazole was started by parenteral administration. The uterus was evacuated under general anaesthesia. After evacuation of the uterus, the vaginal bleeding continued, the pyrexia did not respond to therapy, and the diarrhoea persisted. The platelet count did not rise above 20 000/μl; although the crude clotting time, prothrombin index and fibrinogen level remained within the normal reference range. On the 4th day after admission a total abdominal hysterectomy was performed and the antibiotic therapy was changed to gentamicin 80 mg 8-hourly. Metronidazole therapy was continued.

Pyrexia and diarrhoea, with passage of blood-stained mucus, continued after the hysterectomy and a sigmoidoscopy was performed on the 2nd postoperative day. A granular rectal mucosa was seen with blood issuing from higher up in the colon; histological examination of a biopsy specimen of mucosa revealed amoebic ulceration. The fever and diarrhoea persisted; gentamicin therapy at adequate levels and metronidazole therapy were maintained, as were supportive fluid and electrolyte therapy. Throughout this period the blood urea levels remained raised, although they were lower than on admission.

On the 11th day after admission the diarrhoea abated, although the patient was still pyrexial. She now complained of restlessness, facial twitching and muscular cramps. Physical examination revealed positive Chvostek and Trousseau signs and increased reflexes; the other systems were essentially normal. The biochemical findings at this stage are set out in Table I. Essentially, the patient was hypocalcaemic, hypomagnesaemic and hypokalaemic. Magnesium excretion in the urine was high in the reference range in spite of the hypomagnesaemia, and serum alkaline phosphatase activity and urinary calcium excretion were within reference range values; the acid-base balance was not disturbed. Parathyroid status was assessed by measuring urinary cyclic adenosine monophosphate (cAMP) response to parathyroid hormone infusion; a 12-fold increase was recorded.

<table>
<thead>
<tr>
<th>TABLE I. BASAL BIOCHEMICAL DATA (REFERENCE RANGES IN BRACKETS)</th>
<th>Patient's values</th>
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</thead>
<tbody>
<tr>
<td>Serum bicarbonate (24 - 32 mmol/l)</td>
<td>26,0</td>
</tr>
<tr>
<td>Serum potassium (3,5 - 5,0 mmol/l)</td>
<td>2,8</td>
</tr>
<tr>
<td>Serum calcium (2,25 - 2,75 mmol/l)</td>
<td>1,62*</td>
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<tr>
<td>Serum phosphate (0,81 - 1,45 mmol/l)</td>
<td>1,44</td>
</tr>
<tr>
<td>Serum albumin (35 - 50 g/l)</td>
<td>25,0</td>
</tr>
<tr>
<td>Serum alkaline phosphatase (30 - 85 μU/ml)</td>
<td>62,0</td>
</tr>
<tr>
<td>Serum magnesium (0,74 - 0,99 mmol/l)</td>
<td>0,35</td>
</tr>
<tr>
<td>24-h urinary calcium (1,25 - 10 mmol/24h)</td>
<td>2,66</td>
</tr>
<tr>
<td>24-h urinary magnesium (3 - 10 mmol/24h)</td>
<td>10,43</td>
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</tbody>
</table>

*Adjusted after applying Payne's correction for hypo-albuminaemia.
On the strength of these findings the gentamicin therapy was withdrawn and cefalothin substituted. Metronidazole therapy was withdrawn 4 days after the diarrhoea stopped, and therapy with oral magnesium chloride (elemental magnesium 0.4 g/d) and intramuscular magnesium sulphate (1.0 g/d) was initiated. The response to magnesium therapy is set out in Fig. 1. By the 10th day the patient had achieved normocalcaemic and normomagnesaemic status. Serum potassium levels were normal at this stage and she was pyrexial 20 days after admission. The patient maintained normal calcium and magnesium blood levels until her discharge; at a follow-up visit 6 weeks later serum calcium, magnesium and potassium levels were normal.

Fig. 1. The response of serum magnesium and calcium levels to magnesium therapy.

Discussion

The combination of amoebic dysentery and puerperal sepsis is an unusual affliction. In this patient it certainly set the stage for potential magnesium loss via the gut in the classic sense: prolonged diarrhoea with supportive fluid and electrolyte therapy without magnesium replacement. The contribution of the diarrhoea to magnesium loss could not be assessed retrospectively because the hypomagnesaemic state was only demonstrated when the diarrhoea stopped, but it is reasonable to assume that it made a contribution. The fact that the patient was on gentamicin therapy during a period when her blood urea level was raised is of interest; it has recently been reported that gentamicin therapy results in urinary magnesium loss.

As the results in Table I show, the patient's urinary magnesium excretion was at the upper limit of the reference range in the presence of hypomagnesaemia. Whereas a cause-and-effect relationship between this observation and the gentamicin therapy cannot be considered as unequivocally established in the patient, the fact that she became normomagnesaemic and remained so until discharge, after gentamicin withdrawal and appropriate replacement therapy, argues strongly for the contention that gentamicin therapy contributed to magnesium depletion.

The effect of magnesium depletion on calcium and potassium metabolism is well documented, although the mechanisms involved are less clearly established. In the patient described here there can be little doubt that the hypomagnesaemia induced a functional hypoparathyroidism. The cAMP response to parathyroid hormone infusion was unequivocal, suggesting hypoparathyroidism. The patient recovered on magnesium therapy alone and, on recovery, maintained normocalcaemia and normomagnesaemia. These findings therefore support the view that hypomagnesaemia can cause functional hypoparathyroidism.

Less clear, from the present report, is the effect of hypomagnesaemia on potassium metabolism. The potassium level reverted to normal with a return to normal calcium and magnesium levels, but it also coincided with the general recovery of the patient and discontinuation of intravenous fluid and electrolyte therapy.

This report emphasizes the importance of monitoring magnesium levels in patients with prolonged diarrhoea on fluid and electrolyte replacement therapy, and the need to be aware of the urinary magnesium loss in patients on gentamicin therapy.

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REFERENCES