were receiving 14 tablets of Slow Sodium (140 mEq sodium) per dialysis. As no alteration in mean blood pressure or body weight was detected it is concluded that Slow Sodium is a useful drug in the prophylaxis of muscle cramps in patients on maintenance haemodialysis for end-stage renal failure.

We wish to thank Mrs. Susan Standen for statistical advice, Ciba pharmaceutical company for the supply of tablets, and the nursing staff of the medical renal unit for their help.

MEDICAL MEMORANDA

Symptomatic Hypomagnesaemia after Parathyroidectomy

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Introduction

Hypomagnesaemia may occur in a number of clinical situations, including states of malnutrition or malabsorption, after the loss of gastrointestinal secretions by continuous aspiration, in chronic alcoholics, and in primary and secondary aldosteronism. Primary hyperparathyroidism may also be associated with low serum magnesium levels (Harmon, 1956) particularly after parathyroidectomy in those patients with extensive bone disease (King and Stanbury, 1970). In this situation hypocalcaemia and hypomagnesaemia co-exist and this is also common in many of the other low magnesium states, so that it has often proved difficult to attribute the clinical picture to a deficiency of one or other cation.

The present patient with primary hyperparathyroidism and generalized bone disease developed profound hypomagnesaemia and hypocalcaemia after parathyroidectomy. These changes were associated with acroparaesthesiae, muscle weakness, tremor, and mental changes, all of which were reversed by restoration of the serum magnesium level to normal despite a persistently low serum calcium level.

Case Report

A 67-year-old woman presented with a pathological fracture of the left femoral neck. There was a five-year history of diffuse bone pains and other symptoms compatible with hypercalcaemia. Radiography showed widespread cystic bone lesions, chondrocalcinosis articularis, and metastatic calcification. The clinical diagnosis of primary hyperparathyroidism was confirmed by the finding of hypercalcaemia (13.0 mg/100 ml), hypophosphataemia (2.2 mg/100 ml), and a raised level of circulating immunoassayable parathyroid hormone of 30 ng/ml (normal below 0.5 ng/ml).

At operation a benign parathyroid adenoma of chief cell type was excised; the remaining glands appeared macroscopically normal.

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Postoperative biochemical changes are shown in the chart. Despite a progressive fall in the serum calcium level, the patient was at first entirely asymptomatic. Oral calcium and vitamin D supplements were started after eight days. On the 10th postoperative day, however, she became strikingly agitated, experiencing episodes of terror and complaining of painful paraesthesiae in the hands and feet. Marked muscle weakness and a generalized fine tremor were observed, but tendon reflexes were normal and Chvostek's and Trousseau's signs remained negative. Serum calcium and potassium levels of 6.0 mg/100 ml and 3.5 mEq/l respectively were recorded at this time. Despite regular oral supplements and additional intermittent intravenous infusions of calcium her symptoms worsened.

Biochemical changes in patient after operation.

On the 14th day when the clinical picture was most striking, two low serum magnesium levels of 0.7 and 0.9 mg/100 ml were recorded at a time when the serum calcium level had reached a nadir at 4.8 mg/100 ml. An intravenous infusion of 200 mg of elemental magnesium as magnesium sulphate in saline was therefore administered over a period of two hours. This produced a dramatic symptomatic improvement; the paraesthesiae, tremor and anxiety state lessened and muscle power improved dramatically. Immediately after the infusion, the serum magnesium level had risen to 2.4 mg/100 ml, the calcium level remaining at 5.5 mg/100 ml. Less severe symptoms had recurred by the next day when she had again become hypomagnesaemic, and oral magnesium as the hydrated oxide (equivalent to 1.7 g elemental magnesium daily) was therefore started. During the subsequent week, as the serum magnesium level rose towards the normal range, there was a progressive and sustained clinical improvement. No change in the serum calcium level occurred until the 22nd day after surgery at which time the serum magnesium had returned to a low normal value of 1.7

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mg/100 ml, there being no recurrence of symptoms. On the 17th postoperative day the serum parathyroid hormone level was 3.3 ng/ml.

The only additional noteworthy event during the recovery period was an episode of acute polyarthritides which rapidly settled with anti-inflammatory analgesics. In view of the previous radiological finding of chondrocalcinosis articularis this was presumed to be a crystal synovitis.

Comment

A variety of symptoms have been attributed to hypomagnesaemia. Harmon (1956) described neuromuscular irritability and convulsions in a child with a parathyroid adenoma, Potts and Roberts (1958) listed mental confusion, muscle weakness with fasciculation, and clonic convulsions in cases of hypomagnesaemia after parathyroidectomy, and Fletcher *et al.* (1960) ascribed weight loss, apathy, muscular weakness, and impairment of memory to magnesium deficiency after massive intestinal resection. Vallee *et al.* (1960) reported five patients in whom tetany associated with positive Chvostek and Trousseau signs responded to magnesium administration and, in an experimental clinical investigation designed to study pure magnesium depletion, Shils (1969) noted anorexia, mental disturbances, and muscle weakness with fasciculation and tremor, but positive Chvostek and Trousseau signs were inconstant.

Associated electrolyte disturbances, particularly hypocalcaemia, often complicate the interpretation of clinical symptoms in hypomagnesaemic states. In our patient, despite persistence of severe hypocalcaemia the acute restoration of the serum magnesium level to normal with intravenous therapy was accompanied by a striking alleviation of the clinical abnormalities, and deterioration was associated with the subsequent return of the serum magnesium to subnormal levels. We conclude that the extreme muscle weakness, tremor, and psychiatric syndrome of pathological anxiety and fear were attributable to profound hypomagnesaemia.

Sustained biochemical improvement was achieved only when oral magnesium supplementation had resulted in a progressive rise in the serum magnesium concentration. Restoration of normal serum calcium levels appeared to be

facilitated by provision of magnesium as has been reported previously in a variety of clinical situations. (Estep *et al.*, 1969, Muldowney *et al.*, 1970). The mechanism whereby magnesium depletion leads to defective calcium homeostasis has been investigated in a variety of ways. Until recently it was believed that hypomagnesaemia interfered with the peripheral response to parathyroid hormone leading to a condition of end-organ refractoriness. But recent work in primary hypomagnesaemia has suggested that parathyroid hormone secretion may be impaired and thus the hypocalcaemia explained on the basis of a conditioned hypoparathyroidism (Se Mo Suh *et al.*, 1973). In our patient on the 17th postoperative day when the serum magnesium was at a subnormal level and the serum calcium still profoundly depressed, the serum immunoreactive parathyroid hormone was 3.3 ng/ml—a level well above normal.

It thus appears that in the postparathyroidectomy state, hypocalcaemia may persist despite high circulating parathyroid hormone levels until normal magnesium concentrations have been restored.

The observations in this patient serve to re-emphasize that magnesium deficiency may make an important contribution to the clinical and biochemical disturbances which follow surgery in primary hyperparathyroidism with bone disease.

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