Hypomagnesaemia causing myopathy and hypocalcaemia in an alcoholic

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Summary: A well-nourished alcoholic patient developed a subacute myopathy which responded rapidly to correction of severe hypomagnesaemia. The finding of profound hypocalcaemia prompted the measurement of serum magnesium. Magnesium deficiency should be looked for in any alcoholic patient with a myopathy as the prognosis seems better than in many other forms of alcoholic myopathy. Correction of the magnesium deficiency corrects the hypocalcaemia without the need for calcium supplementation.

Introduction

The cause of subacute alcohol associated myopathy is not always clear. Hypokalaemia has been implicated¹ but clinical response to correction of hypokalaemia does not always occur. We describe a hypomagnesaemic hypocalcaemic normokalaemic patient with such a myopathy that responded well to magnesium supplementation.

Case report

A 65 year old well-nourished woman presented with a 6 week history of muscle cramps and arm and leg weakness predominantly affecting proximal musculature. She had a long history of excessive alcohol intake and had suffered a short episode of similar weakness 1 year previously which had resolved spontaneously. She took frusemide 40 mg a day for intermittent dependant oedema and was taking potassium supplements. Proximal muscle weakness was pronounced in both arms and legs and there was some muscle twitching but no fasciculation. Chvostek's sign was positive but Trousseau's sign was negative. Although deep tendon reflexes were absent there were no other features of peripheral neuropathy.

Investigations revealed the following serum abnormalities (normal ranges in brackets): aspartate transaminase 79 IU/l (normal 5–30), albumin 30 g/l (38–46), bilirubin 27 μ mol/l (3–21), calcium 1.35 mmol/l (2.10–2.55) and magnesium 0.20 mmol/l (0.70–0.95). Alkaline phosphatase remained between 200–220 IU/l (70–350) throughout the illness. Serum potassium and

creatinine kinase levels were also normal. Electromyography (EMG) showed myopathic change and sensory action potentials were normal. Muscle biopsy (vastus lateralis) showed scattered atrophic muscle fibres with no group atrophy and no evidence of myositis.

Serum magnesium was measured within hours of admission to hospital after serum calcium had been found to be profoundly low. The hypocalcaemia was thought to be secondary to the hypomagnesaemia² and attributable predominantly to alcoholism and partly diuretic therapy. Replacement of the magnesium deficit and withdrawal of alcohol and diuretics resulted in a gradual return of serum calcium concentration to normal (Figure 1). Neuromuscular irritability resolved over the first 2 days; the proximal muscle weakness taking about 2 weeks to recover. Deep tendon reflexes remained absent. Following treatment the serum calcium was 2.2 mmol/l, serum magnesium 0.92 mmol/l and serum phosphate 1.09 mmol/l (normal 0.8–1.4).

Discussion

Hypomagnesaemia may cause hypocalcaemia and under these conditions attempts at correcting the hypocalcaemia with calcium supplements and/or vitamin D, even in massive doses, are futile.² The treatment of this form of hypocalcaemia requires administration of magnesium rather than calcium – a point of management that makes hypomagnesaemia a very important differential diagnosis in any patient with unexplained severe hypocalcaemia. Recognition

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Figure 1 Changes in serum magnesium and calcium with magnesium supplementation.

of this fact avoids unnecessary and futile administration of calcium supplements and vitamin D. The pathogenesis of this secondary hypocalcaemia has been reviewed by Berkelhammer³ and Rude⁴ and briefly involves both a decrease in parathyroid hormone (PTH) secretion and a peripheral resistance to PTH action, although impaired 25-hydroxylation of vitamin D may also be involved.⁵ Correction of the magnesium deficit results in resolution of the hypocalcaemia over a few days as illustrated by our patient (Figure 1). We also demonstrate that serum calcium concentrations do not closely follow acute changes in serum magnesium concentrations suggesting that intracellular magnesium levels are more important in influencing calcium homeostasis. Our patient was given more than 750 mmol of magnesium ions over 3 weeks to correct the hypomagnesaemia. This emphasizes the fact that, as with potassium, magnesium is mainly an intracellular element. Consequently patients may have significant depletion of body magnesium even when the serum magnesium concentration is normal.⁵

Hypomagnesaemia is well recognized as a complication of alcoholism and is thought to be secondary to decreased intake, reduced absorption and excessive renal losses.⁶ Magnesium depletion is not generally recognized as a possible cause of alcoholic myopathy. Cases of alcoholic myopathy have been attributed to hypokalaemia even when there has been associated hypomagnesaemia and when clinical improvement

has followed treatment with both potassium and magnesium supplements.⁷ There is some evidence that muscle may be at particular risk of magnesium depletion. For instance Lim & Jacob⁸ showed reduction in muscle magnesium in alcoholics who had normal bone magnesium levels and McIntyre⁹ has shown the same to occur in magnesium deficiency due to severe malabsorption. Experimental depletion of magnesium in animals causes alteration in muscle resting transmembrane electrical potential¹⁰ and even muscle necrosis.¹¹ In normal human volunteers such depletion causes myopathic changes on EMG which resolve with magnesium repletion.² Fonseca & Havard¹² have reported a patient with cardiac failure secondary to hypomagnesaemia and in the absence of arrhythmias. Cardiac muscle with its similarities to skeletal muscle may have expressed a myopathy in a similar manner to our patient's skeletal muscle myopathy. However, the patient they reported was so severely malnourished that various other nutritional deficiencies may have contributed to the clinical picture.

Our patient shows that subacute alcoholic myopathy may be due to magnesium deficiency and may show good response to magnesium repletion. Although significant magnesium depletion may occur in the absence of hypomagnesaemia or hypocalcaemia, we suggest that measurement of serum magnesium concentration should form part of the investigation of every alcoholic patient with a myopathy.

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