A Case of Hypermagnesemia Accompanied by Hypercalcemia Induced by a Magnesium Laxative in a Hemodialysis Patient

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Dear Sir,

It is understood that hypermagnesemia can occur in patients with chronic renal failure during the administration of magnesium even at a pharmacological dose. According to previous reports about hypermagnesemia, which the authors could refer to, the serum calcium level of those patients was decreased or unchanged [1,2].

We report our experience with a case of hypermagnesemia accompanied by hypercalcemia induced by a magnesium laxative, which occurred in a hemodialysis patient. A 63-year-old male was transferred to the Kita Hospital from a dialysis clinic because of neurological changes. He suffered from chronic renal failure and anuria due to chronic glomerulonephritis, having received hemodialysis for 7 years. The patients had had occasional episodes of obstipation. 6 weeks previously, he had complained of a sense of fullness, loss of appetite, and no passage for 10 days. Magnesium oxide powder was administered at 3.0 g/day, since different purgatives tried up to then had failed to improve his complaint. A few days later, he had a passage every day, and his appetite improved. In the 4th week, however, he began to manifest vomiting, became irritable, confused, and disoriented regarding time and surroundings.

On admission, he was of medium stature for a Japanese male: 160 cm in height, weighing 54 kg. His state of consciousness was stuporous. However, deep tendon reflexes were present and the respiratory movement was not impaired. Blood pressure was 216/104 mm Hg. Pulse rate was 80, regular and full. The ECG showed a regular sinus rhythm. The blood chemistry revealed a marked elevation of serum magnesium up to 9.9 mg/dl. This was on the 35th day, since magnesium administration was begun. We decided to start dialyzing every day. His state of consciousness improved on the 4th day. Intermittent hemodialysis was performed every day until the 23rd hospital day, when the serum magnesium level returned to normal.

Looking back over the course of the 231 days (see fig. 1), from 77 days before until 154 days after this patient received magnesium, at what drugs were taken, how many times dialysis was received, and the blood chemistry of the patient, we noticed the following. (1) Serum total magnesium, which had been at the level of 2.6-3.3 mg/dl previously, began to increase 2 weeks after the initiation of the administration, and total calcium, 8.5-9.9 mg/dl initially, also began to increase parallel with magnesium, reaching its peak serum level of 13.5 mg/dl. (2) A remarkable decrease of the serum phosphate level (7.9 mg/dl initially to 1.7 mg/dl 28 days after administration) and alkaline phosphatase level (from 176 to 42 Babson units/1105 days after administration) occurred after administering magnesium. (3) During the overall course, 1 α -(OH)D₃ (1 α -hydroxycholecalciferol) was administered at the dose of 1.5 μ g/ day. (4) There was no administration of calcium salts.

Serum calcium elevation appeared to occur simultaneously with that of magnesium. This implies that the administration of magnesium might have played a role as a trigger to elevate the serum calcium level. This phenomenon can happen, as many authors have already described, in cases of hypocalcemic and hypomagnesemic states, in which not only the supplementation of calcium and vitamin D₃, but also the administration of magnesium are necessary for restoring the serum calcium level [3-5]. To our regret, we failed to evaluate the serum PTH level or 1,25-(OH)₂ vitamin D. Our patient was probably in a hyperparathyroid state, considering the high level of serum alkaline phosphatase, which was about 2- to 5-fold above the normal value, and not in a hypo-parathyroid state. After the administration of magnesium, the parathyroid function might have been suppressed, and serum alkaline phosphatase become low. On the other hand, he was not obviously hypomagnesemic initially. However, it could be possible that the intracellular magnesium level had actually been low, as Lim et al. [6] had described, and changing of serum magnesium to a higher level, by adding large amounts of magnesium oxide powder, might have been activated the pharmacological effect of 1 α -(OH) vitamin D₃ on calcium metabolism, resulting in hypercalcemia. This case suggests that some mechanisms exist in magnesium-calcium metabolism, which are not well defined yet.

HD every day

HD 3 times a week HD 3 times a week

Mg (1.5-3.0 mg/dl) Ca (8.7-10.1 mg/dl) P (2.5-4.5 mg/dl)

150 200 days

160

120

Serum alkaline phosphatase (9-35 units/I)

50 100 150

MgO 3 g daily

Fig. 1. Medications given to this patient, including the dialysis sessions and drugs. All drugs were administered orally. Line graphs indicate changes in serum total magnesium, total calcium, phosphate and alkaline phosphatase level. The alkaline phosphatase level is given in Babson units/l. Blood chemistry was examined approximately every 5-14 days at the initiation of every dialysis session. HD = Hemodialysis.

-100 -50 0

200 days

1 α -OH-D₃ 1.5 μ g daily

Other drugs (daily): se[®]nosides 72 mg, dried aluminum hydroxide gel 3.0 g, nicardipine hydrochloride 120 mg, hydralazine hydrochloride 150 mg, allopurinol 200 mg

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