

Casos clínicos

Severe hypocalcemia secondary to hypomagnesaemia, successfully treated by self-administered subcutaneous magnesium

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Abstract

We report the case of a patient with recurrent severe hypocalcemia, secondary to hypomagnesaemia, and pre-renal renal failure, due to ileostomy losses after a colectomy, who needed several admissions to the hospital through more than one year. Finally, he was successfully treated by self-administered subcutaneous magnesium: he reached and maintained normal levels of serum calcium, magnesium and PTH, no more hospital admission were needed and he resumed a normal life.

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Key words: *Hypocalcemia. Hipomagnesaemia. Self administration. Magnesium.*

Introduction

Hypocalcemia is an important clinical entity which causes abnormal neurological sensations and neuromuscular hyperexcitability.¹ One of the causes of hypocalcemia is hypoparathyroidism, which can be secondary to hypomagnesaemia.¹ Magnesium deficiency due to gastrointestinal disease is usually treated with oral supplements. Some patients who do not need parenteral nutrition suffer chronic and recurrent fluid and electrolyte depletion despite oral supplementation,¹ so that they need parenteral magnesium supplementation. We report the case of a patient with recurrent severe hypocalcemia secondary to hypomagnesaemia

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HIPOCALCEMIA SEVERA, SECUNDARIA A HIPOMAGNESEMIA, TRATADA CON ÉXITO MEDIANTE AUTO-ADMINISTRACIÓN SUBCUTÁNEA DE MAGNESIO

Resumen

Se trata de un paciente con hipocalcemia severa recurrente, secundaria a hipomagnesaemia, y fracaso renal agudo prerrenal secundario a pérdidas digestivas a través de una ileostomía, tras una colectomía. Por este motivo necesitó varios ingresos hospitalarios a lo largo de más de un año. Finalmente fue tratado con éxito mediante auto-administración subcutánea de magnesio. Con este tratamiento mantuvo niveles normales de calcio, magnesio y PTH, no requirió más ingresos hospitalarios y recuperó una vida normal.

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Palabras clave: *Hypocalcemia. Hipomagnesaemia. Auto-administración. Magnesio.*

and pre-renal renal failure successfully treated by self-administered subcutaneous magnesium.

Case report

A 71-year-old man, former smoker, with past medical history of hypertension, hyperlipidemia and surgery for an abdominal aortic aneurysm, was diagnosed of rectum carcinoma and treated by chemotherapy and radiotherapy prior to an anterior resection of rectum with colorectal anastomosis and protection ileostomy. After surgery he was admitted to the hospital twice because of pre-renal acute renal failure secondary to ileostomy losses, and successfully treated by intravenous fluid replacement.

Nine months after rectum surgery, a right and transverse colectomy was performed because the patient had colonic polyps, and a postsurgical ischemic cholangitis led to a left colectomy and a terminal ileostomy. He had a pre-renal acute renal insufficiency secondary to high ileostomy losses after this second surgery.

Table I
Clinical and biochemical evolution of the patient before subcutaneous magnesium treatment

Weeks after anterior resection of rectum	4	40	52	55	59
Clinical events	Acute renal insufficiency	Total colectomy and terminal ileostomy. Acute renal insufficiency	Admission. Vomits and fever. Seizures. Admission on ICU	Admission. Fever, tremor, asthenia	Admission. Asthenia. Tremor. Subcutaneous magnesium was initiated during this admission
Total serum calcium (mmol/l) Normal range: 2.15-2.55			1.55	1.68	1.58
Ionized calcium (mmol/l) Normal range: 1.15-1.29			0.63	0.8	0.91
Serum magnesium (mmol/l) Normal range: 0.66-1.07			0.42	0.08	0.21
PTH (pg/ml) Normal range: 10-65			26	44	57
Creatinine (μ mol/l) Normal range: 62-106	504	636	256	230	194

Three months after the colonic resection he was again admitted to the hospital due to vomiting and fever. Five days after admission he was transferred to the Intensive Care Unit because he had seizures secondary to severe hypocalcemia and acute renal failure. He was discharged on treatment with loperamide, oral calcium and calcitriol, but had to be readmitted one week later because he had severe hypocalcemia secondary to hypomagnesaemia and prerenal acute renal failure. He was treated with intravenous infusion of fluids, calcium and magnesium and he was discharged. Oral supplements of magnesium, codeine and octreotide were added to the previous treatment. He came back two weeks later (five months after colectomy): he reported tremor and intense discomfort and was admitted again because of severe hypocalcemia, hypomagnesaemia and acute renal insufficiency. The first day he was treated with intravenous infusion of saline solution, calcium chloride and magnesium sulfate, and the second day was transferred to subcutaneous administration of saline solution with magnesium sulfate (12 mmol of magnesium sulfate in 1,000 ml of saline solution during 12 hours) via a butterfly needle. The patient and his wife were instructed about self-administered subcutaneous fluid infusion and he was discharged three days after admission with the following daily treatment: oral calcium gluconate, calcitriol, codeine and a self-administered subcutaneous infusion of 500 ml of saline solution with 6 mEq of magnesium sulfate during 6 hours per day. He was advised to increase saline administration to 1,000 ml per day if diuresis diminished or ileostomy losses increased.

After this admission patient has had normal levels of serum calcium, magnesium and PTH. Hospital admis-

sion has not been needed and renal function has remained stable. The patient, whose daily activities had been very limited since colectomy, has resumed a normal life.

Table I summarises clinical and biochemical evolution before subcutaneous magnesium treatment. Table II summarises biochemical evolution after this treatment.

Discussion

Hypocalcemia can be caused by hypoparathyroidism, PTH resistance, vitamin D deficiency, medications, congenital causes, malignancy, and some severe diseases. Hypomagnesaemia causes both hypoparathyroidism and PTH Resistance.¹ Causes of hypomagnesaemia include gastrointestinal losses (including acute or chronic diarrhoea, malabsorption and steatorrhea, or short bowel syndrome), renal losses (including diuretics, volume expansion, alcoholism, hypercalcemia, nephrotoxins and loop of Henle or distal tubule dysfunction)³ and intracellular redistribution (including refeeding syndrome).

Symptoms of hypocalcemia include abnormal neurological sensations and neuromuscular excitability and central nervous system manifestation like seizures, mental status changes and irritability.¹ Hypomagnesaemia is usually asymptomatic but it can produce neuromuscular irritability, hypokalemia and cardiac arrhythmia.⁴ Seizures secondary to hypomagnesaemia in gastrointestinal diseases have been previously reported.⁵

Our patient's hypocalcemia was secondary to hypoparathyroidism, and maybe PTH resistance, secondary

Table II
Biochemical evolution of the patient after subcutaneous magnesium treatment

Days after discharge	2	5	9	16	35	68	98	137
Total serum calcium (mmol/l) Normal range: 2.15-2.55	2.15	2.37	2.35	2.42	2.36	2.45	2.48	2.45
Serum magnesium (mmol/l) Normal range: 0.66-1.07	0.71	0.76	0.76	0.67	0.76	0.71	0.67	0.71
PTH (pg/ml) Normal range: 10-65	111	70	49	49	34	28	31	40
Creatinine (μ mol/l) Normal range: 62-106	133	124	124	133	115	115	106	124

to hypomagnesaemia, since his PTH level was low or inappropriately normal for his hypocalcemia, and not high, as it is expected in non-PTH related hypocalcemia.

Our patient's high-output ileostomy was responsible for the repeated episodes of dehydration and acute renal failure, in a similar way as it happens in chronic diarrhoea. We think it was also responsible for hypomagnesaemia, that was worsened by the absence of the colon, an organ with magnesium-absorbing capacity.⁴ The remarkable improvement with subcutaneous administration of a dose of magnesium close to the 10 mMol usually absorbed per day through the gastrointestinal tract⁴ reinforce this etiology, since this treatment would not have corrected a hypomagnesemia originated from renal magnesium wasting.

Hypocalcemia was resistant to treatment with oral calcium and calcitriol (consistently with diagnosis of hypocalcemia secondary to hypomagnesaemia) and oral supplements of magnesium were not effective. Self-administered subcutaneous fluid and magnesium infusion was an effective alternative, avoided new admissions and improved the quality of life of our patient.

Previous reports of self-administered subcutaneous MgSO₄ infusion are scarce and authors emphasize the usefulness and easiness of this treatment.² Our experience supports that it is a practical and safe method of

managing hydroelectrolytic deficiencies in patients who do not respond to oral treatment.

Conclusions

Hypomagnesaemia secondary to gastrointestinal diseases can produce hypoparathyroidism and severe hypocalcemia with seizures and other complications. Self-administered subcutaneous fluid and magnesium infusion is an effective alternative in patients with malabsorption or increased losses who do not respond to oral treatment and do not need total parenteral nutrition.

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