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CASE REPORT

Recurrent Hypocalcaemic Spasms in an Elderly Woman

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Abstract

Hypocalcaemia is common in elderly patients and is usually attributed to vitamin D deficiency or inadequate calcium intake. Recurrent symptomatic hypocalcaemia despite long-term supplementation is uncommon and suggests an underlying metabolic abnormality. We report a 75-year-old woman with recurrent hypocalcaemic spasms over two years, with increased frequency over three months, requiring repeated intravenous calcium therapy. Despite prolonged oral calcium and vitamin D supplementation, she continued to develop symptoms. Biochemical evaluation revealed low-normal parathyroid hormone levels, normal renal function, and clinical response to magnesium during acute episodes. A diagnosis of functional hypoparathyroidism secondary to magnesium deficiency was considered. Treatment with calcium citrate, calcitriol, and oral magnesium resulted in sustained clinical improvement. This case highlights the importance of evaluating magnesium status in elderly patients with refractory hypocalcaemia.

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INTRODUCTION

Hypocalcaemia is frequently encountered in clinical practice, particularly among elderly patients. Common causes include vitamin D deficiency, chronic kidney disease, hypoparathyroidism, and malabsorption. Most patients respond to oral calcium and vitamin D supplementation. However, recurrent symptomatic hypocalcaemia despite adequate therapy is uncommon and warrants evaluation for alternative mechanisms. Magnesium plays a critical role in calcium homeostasis by regulating parathyroid hormone secretion and action. Magnesium deficiency can lead to functional hypoparathyroidism, resulting in refractory hypocalcaemia. [1, 2] We present a case highlighting this often-overlooked mechanism in an elderly patient.

CASE REPORT

A 75-year-old woman presented with recurrent painful carpopedal spasms for two years, with five episodes occurring over the preceding three months. Each episode required emergency treatment with intravenous calcium gluconate. She had a history of hypertension, stable angina with minimal coronary artery disease, lumbar spondylosis, and long-standing bronchial asthma. She had been receiving oral calcium carbonate 500 mg once or twice daily along with vitamin D3 supplementation for approximately five years.

There was no history of thyroid and parathyroid malignancy or surgery, chronic kidney disease, malabsorption, or seizures. Physical examination between episodes was unremarkable. Laboratory evaluation showed normal renal function, serum magnesium of 1.9 mg/dL and serum intact parathyroid hormone of 19.62 pg/mL, which was inappropriately low-normal in the context of hypocalcaemia. Spot urinary calcium was 13.3 mg/dL. During acute episodes, symptomatic improvement was consistently observed when magnesium was co-administered with intravenous calcium.

Based on clinical and biochemical findings, functional hypoparathyroidism secondary to magnesium deficiency was considered. The patient was started on calcium citrate 500 mg twice daily, calcitriol 0.25 microgram once daily, and oral magnesium 200 mg twice daily supplementation, following which she remained asymptomatic. [1, 2, 5]

REVIEW OF LITERATURE

Magnesium is an essential divalent cation involved in numerous enzymatic reactions and plays a pivotal role in calcium homeostasis. Hypomagnesaemia is a recognised but frequently underdiagnosed cause of hypocalcaemia, particularly in elderly patients. Magnesium deficiency affects calcium balance by impairing both the secretion of parathyroid hormone and its action at target organs, leading to a state of functional hypoparathyroidism. [1, 2]

Elderly individuals are especially vulnerable to magnesium deficiency due to reduced dietary intake, impaired gastrointestinal absorption, renal losses, and polypharmacy. Commonly used medications such as diuretics and proton pump inhibitors may further exacerbate magnesium depletion. [3] In such patients, hypocalcaemia may persist despite adequate calcium and vitamin D supplementation unless magnesium deficiency is corrected.

Several studies have demonstrated that hypocalcaemia refractory to calcium therapy responds only after magnesium repletion. Rude and colleagues described magnesium deficiency as a cause of heterogeneous disease manifestations, including neuromuscular excitability and tetany. [1] Agus highlighted that magnesium deficiency can result in both decreased parathyroid hormone secretion and peripheral resistance to parathyroid hormone, producing biochemical features similar to hypoparathyroidism. [2]

The choice of calcium preparation is also relevant in elderly patients. Calcium carbonate requires an acidic gastric environment for optimal absorption, which may be reduced in older individuals. Calcium citrate is absorbed independently of gastric acid and has superior bioavailability in elderly patients and those on long-term medications. [4] Active vitamin D analogues such as calcitriol bypass the need for renal activation and are beneficial in patients with impaired vitamin D metabolism or functional hypoparathyroidism. [4,5]

Indian studies have emphasised the need to evaluate secondary causes of hypocalcaemia, including magnesium deficiency, particularly in patients with recurrent symptoms. Failure to recognise this entity can lead to repeated hospitalisations, unnecessary escalation of calcium therapy, and persistent morbidity.

DISCUSSION

This case highlights an important and often overlooked cause of recurrent hypocalcaemia in elderly patients. Despite long-term calcium and vitamin D supplementation, the patient continued to experience recurrent hypocalcaemic spasms requiring intravenous therapy. The low-normal parathyroid hormone level in the setting of hypocalcaemia suggested an inadequate physiological response rather than classical hypoparathyroidism. [5]

Magnesium deficiency results in impaired parathyroid hormone secretion and reduced responsiveness of bone and kidney to parathyroid hormone, leading to persistent hypocalcaemia. In such cases, calcium supplementation alone is ineffective unless magnesium deficiency is corrected. The patient's consistent clinical improvement following magnesium administration during acute episodes strongly supports this mechanism. [1, 2]

Another contributing factor may have been reduced calcium absorption from long-term use of calcium carbonate in an elderly individual. Calcium citrate offers superior absorption independent of gastric acidity and is therefore preferred in this age group. Additionally, the use of calcitriol ensured adequate intestinal absorption of calcium and bypassed potential defects in vitamin D activation. [4,5]

Recognition of functional hypoparathyroidism secondary to magnesium deficiency is clinically important, as it is readily reversible with appropriate therapy. Early diagnosis and targeted treatment can prevent recurrent emergency visits,

reduce healthcare burden, and improve patient quality of life. [1–3, 6]

CONCLUSION

Recurrent hypocalcaemic spasms in elderly patients despite adequate calcium and vitamin D supplementation should prompt evaluation for magnesium deficiency and functional hypoparathyroidism. Correction with calcium citrate, calcitriol, and magnesium supplementation can result in sustained clinical improvement and prevent recurrence.

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Conflict of Interest
Nil.

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Patient identity was concealed, and written informed consent was obtained from the patient for publication of this case report.

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