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Case Report

Recurrent Severe Spasmophylia from Hypocalcaemia: A Case Report

Rekurensi Spasmofilia Berat Akibat Hipokalsemia: Sebuah Studi Kasus

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ABSTRACT

Hypocalcemia can present with a variety of clinical symptoms and indications. In situations of nerve-related hypocalcemia, muscle cramps and tetany are signs of increased excitability in either peripheral nerves or the central nervous system. Here, we described the case of a 58-year-old woman experiencing ongoing pain and cramping in both her upper and lower extremities. Through careful physical, neurophysiological, and radiological examinations, we were able to identify hypocalcemia as the primary cause of her severe muscle spasms and cramps, known as spasmophilia. Further detailed history-taking revealed a past total thyroidectomy. Administration of adequate calcium supplementation successfully relieved the symptoms.

Keywords: *Calcium, hypocalcemia, spasmophilia*

ABSTRAK

Hipokalsemia dapat muncul dengan berbagai gejala dan indikasi klinis. Pada kejadian hipokalsemia pada saraf, gejala kram otot dan tetani merupakan hasil dari overeksitabilitas saraf tepi atau sistem saraf pusat. Dalam studi kasus ini, kami melaporkan kasus seorang wanita berusia 58 tahun yang mengalami nyeri dan kram otot secara terus-menerus pada kedua ekstremitas atas dan bawah. Setelah dilakukan pemeriksaan fisik, pemeriksaan neurofisiologis, dan radiologi dengan seksama, kami mengidentifikasikan hipokalsemia sebagai penyebab utama spasme dan kram otot (spasmofilia) berat yang terjadi. Penggalian riwayat lebih dalam menunjukkan adanya riwayat tiroidektomi total di masa lalu. Suplementasi kalsium yang memadai dapat meringankan gejala.

Kata Kunci: Hipokalsemia, kalsium, spasmofilia

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INTRODUCTION

In the outpatient setting, severe muscle spasms, also known as spasmophilia, pose considerable challenges. These muscle spasms often result from hypocalcemia, a condition characterized by abnormally low levels of calcium. Hypocalcemia can stem from several clinical conditions, with hypoparathyroidism emerging as the most common cause (1,2). Hypoparathyroidism can be either inherited or acquired following a surgery that reduces the functions of the parathyroid gland (3). Manifesting with a wide spectrum of clinical symptoms and signs, hypocalcemia commonly leads to fatigue, numbness, and muscle spasms (4). In severe cases, it can lead to arrhythmias and seizures. Hypocalcemia can be life-threatening if it is not detected and treated promptly (2). In this case report, we present a case of recurrent severe spasmophilia induced by hypocalcemia in outpatient settings at Bethesda Hospital Yogyakarta.

CASE REPORT

A 58-year-old female patient was sent to our department after experiencing muscle cramps, spasms, and pain in both upper and lower extremities for more than six months. She didn't show any signs of weakness or a problem with her gait. An extensive physical examination revealed no abnormalities. Except for Trousseau's and Chvostek's signs, the neurological examination revealed no signs of aberrant muscle tone or uncontrollable movements. She had undergone a total thyroidectomy eight years prior for papillary thyroid carcinoma and started using a calcium-based medicine to raise her calcium levels after that. Other than this, she had no prior history of tumors, malignancies, diabetes, high blood pressure, injuries, or other illnesses.

Prior to her transfer, she underwent a cervical spine X-Ray to evaluate her cervical lesion. As there was no conclusive finding from the X-Ray, she was provided with pain control medications. However, there was no improvement following the administration of gabapentin and magnesium supplementation for her cramps. Laboratory findings showed low serum calcium and high phosphate levels, which suggested severe hypocalcemia, along with low parathyroid hormone levels, which indicated hypoparathyroidism. The level of magnesium was within the acceptable range. The patient received enough thyroid hormone replacement, resulting in a euthyroid state. There was no evidence of myopathy., as her creatinine kinase (CK) and lactate dehydrogenase (LDH) levels remained within normal parameters. Specifically, the parathyroid hormone level was measured at 1.2pg/mL (normal range: 15-65), calcium level was 6.2mg/DL (normal range: 8.6-10.2), and vitamin D level was insufficient (18ng/ mL). Vitamin D and intensified calcium supplementation (2000mg per day) improved the symptoms. The occurrence of further cramps and spasmophilia was prevented. While her calcium level increased, it remained below the normal level (7.8mg/dL).

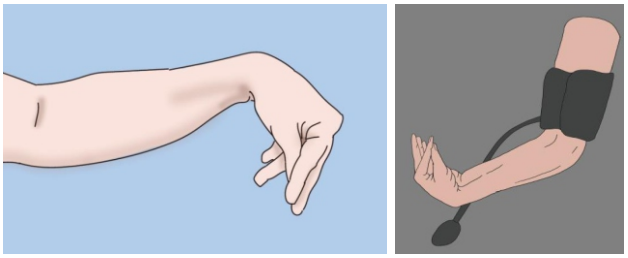

Figure 1. Positive Trousseau's sign (5)

Nerve-related hypocalcemia presents as muscle cramps and tetany , and can be detected with positive Trousseau's signs on examination as were seen in our patient (Figure 1). The sign is observable as a carpopedal spasm that induced by ischemia secondary to the inflation of a sphygmomanometer cuff, commonly on arm, to 20mmHg over their SBP for 3 minutes.

CT of the brain is the best modality for imaging

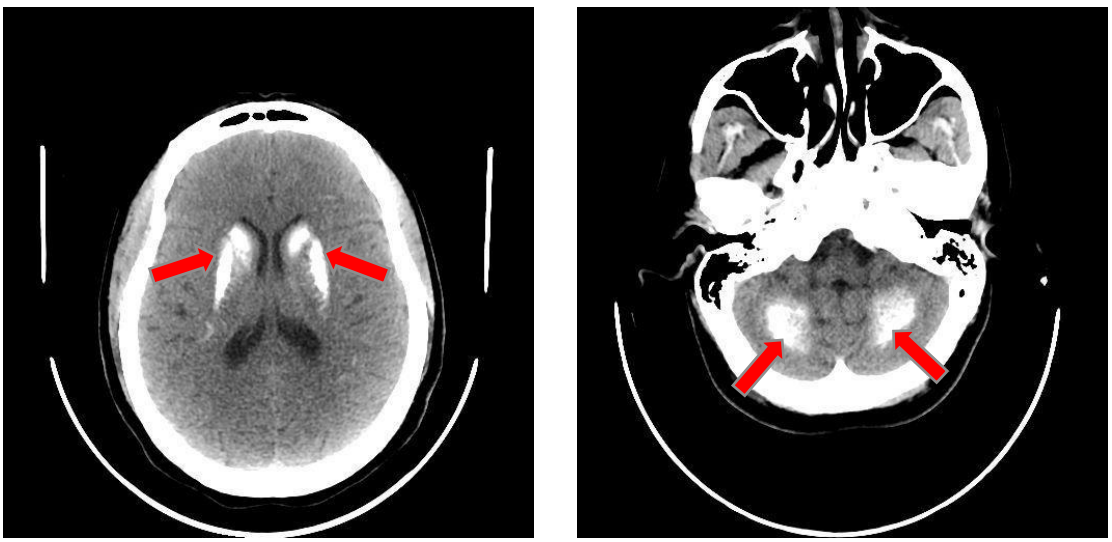

Figure 2. Brain computed tomography (CT) scans demonstrate dense calcification of the bilateral lenticular nuclei (left) and cerebellum (right)

calcification. Brain CT of our patient was shown in Image 2. Dense calcification was found in bilateral lenticular nuclei at the level of roof lateral ventricle. Brain CT also revealed bilateral calcification of the cerebellum at the level of mid-brain (Figure 2).

DISCUSSION

We presented a case study concerning spasmophilia caused by hypocalcemia. Additional tests suggested that hypoparathyroidism was the most likely reason. Both inherited and acquired factors can lead to hypoparathyroidism. The acquired type frequently results from the removal of the parathyroid gland during neck procedures (4,6).

Clinical signs of hypoparathyroidism include circumoral numbness, paresthesia, and muscle spasms affecting the wrists and feet. These symptoms are usually brought on by hypocalcemia. Laryngeal spasms, tetany, and seizures may be present in extreme situations (6,7,8). When calcium concentrations are low, sodium can enter nerve cells, enhancing their sensitivity and causing muscle fasciculations and spontaneous spasms. Imbalances in calcium, magnesium, or potassium concentrations can be the cause of muscle cramps and tetany, which are manifestations of peripheral nerve overexcitability (3,4), Chvostek's sign, which is the twitching of the upper lip upon tapping the cheek along the course of the facial nerve, and Trousseau's sign, the induction of painful carpal spasm by inflating the sphygmomanometer cuff to 20 mm Hg above systolic blood pressure for three minutes are significant physical examination findings (9,10).

In our patient, the positive Trousseau's sign revealed the diagnosis of hypocalcemia (7,9). Further evidence was provided by brain CT scan and laboratory examination, all of which supported the diagnosis of severe hypocalcemia. To diagnose hypoparathyroidism, laboratory evaluation should include the measurements of serum calcium (both total and ionized), serum albumin, phosphate, magnesium, creatinine, intact parathyroid hormone (PTH), and 25-hydroxyvitamin D levels (1,2). In our case, the serum calcium levels were low, and PTH levels were inappropriately low. To exclude deficiency of vitamin D as a potential cause of hypocalcemia, it is crucial to measure 25-hydroxyvitamin D levels. Measurement of magnesium is equally important as magnesium depletion can also lead to hypocalcemia. Our patient was diagnosed with hypoparathyroidism based on low serum calcium (ionized and total), very low PTH, and high phosphate levels.

The presence of severe calcification in either supra tentorial or infratentorial region in our patient supports the diagnosis of hypocalcemia. Intracranial calcification, most usually affecting the bilateral basal ganglia, is a radiological sign of hypoparathyroidism. Also susceptible to calcification include the cerebellum, subcortical white matter, corona radiata, and thalamus (11-13). Hypoparathyroidism and pseudohypoparathyroidism are the two most prevalent pathogenic causes of basal ganglia calcification. Fahr's syndrome, Cockayne's syndrome, mitochondrial illness, tuberous sclerosis, familial idiopathic basal ganglia calcification, Down's syndrome, diffuse neurofibrillary tangles with calcification, , and post-infectious factor are all causes of basal ganglia calcification (11,14).

Suplementation with vitamin D or 1,25 $(OH)_2D_3$) (calcitriol) and combination with a high dose of calcium can be given as a treatment of hypoparathyroidism. A recent meta-analysis showed that taking calcium and vitamin D3 supplementation routinely is related with a decreased risk of hypocalcemia in patients undergoing thyroid surgery (15). For most of the patients, a daily dose of 5000-10000IU of vitamin D and elemental calcium in a dose of ≥1gm/day is usually satisfactory (12,16).

Calcitriol is given in a wide range of doses (0.25 - 2.0g/day). Calcitriol is the most active form of vitamin D metabolite, with a rapid onset of action. For individuals with hypoparathyroidism, calcitriol is the main choice of therapy for vitamin D deficiency (17). Thiazide diuretics, which increase renal calcium reabsorption , may be required to reach a urinary calcium level of 4mg/kg/day. Thiazide also reduces the occurrence of hypocalciuria, which is the cause of kidney stone formation in hypoparathyroid patients who receive long-term calcium and vitamin D supplementation (16,18). In patients with insufficient 25(OH) levels, cholecalciferol may be added to the treatment regimen (16). Calcium doses in patients with chronic hypoparathyroidism are usually 1-3 g of oral elemental calcium divided into 3-4 doses. Calcium citrate and calcium carbonate are the most commonly used forms of administration. For best absorption, calcium carbonate should be taken during or after meals and should not be given simultaneously with other drugs, e.g. L-thyroxine. Calcium citrate offers the benefit of optimal absorption regardless of food consumption (15).

In the case of our patient, oral administration of calcium and vitamin D alleviated all the symptoms. Oral vitamin D and calcium will restore the overall calcium-phosphate balance but do not reverse hypercalciuria seen in hypoparathyroidism (10). Avoiding hypercalciuria and keeping the calcium phosphate product below $55mg^2/dL^2$ ($4.4mmol^2/L^2$) are two of the main goals of long-term management of hypocalcemia, also prevent any complications like nephrolithiasis and other extra-skeletal calcifications (19). A regular biochemical checkup is advised every six months in order to ensure a successful course of treatment (20).

ETHICS

Patient confidentiality has been strictly maintained, with no disclosure of the patient's identity. Written informed consent has been obtained from the patient for publication of the submitted article and accompanying images.

CONFLICT OF INTEREST

We declare that all authors involved in this work have no conflicts of interest. No financial support was provided for the conduct, preparation, data collection, analysis, interpretation, and writting of this report.

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