

Delirium Caused by Hypercalcemia in Older Adults

© Funda Datlı Yakarıılmaz¹, © Zeynel Abidin Öztürk²

¹Malatya İnönü University Faculty of Medicine, Department of Internal Medicine, Malatya, Turkey

²Gaziantep University Faculty of Medicine, Department of Geriatrics, Gaziantep, Turkey

Abstract

Delirium; can be described as a temporary and reversible brain dysfunction syndrome, which manifests itself primarily with physical, physiopathological and psychological disorders. The leading causes of delirium are pneumonia, cancer, urinary infection, electrolyte imbalance (hypo-hyponatremia, hypo-hypercalcemia, hypo-hypermagnesemia), dehydration, congestive heart failure, uremia and stroke. We present a 68-year-old male patient was admitted to the geriatric outpatient clinic with complaints of fatigue, abdominal pain, loss of appetite and weight loss that started about 4 weeks previously. His physical examination was conscious, awake, co-operative but time orientation was not complete. The delirium assessment scale (DAS) was used for delirium evaluation and MDAS 13/30 (delirium >11/30) was detected. At the end of the evaluation the patient was careless and deterioration was detected in the recall test of three items. During the first 48 hours of the treatment, the patient's serum calcium level regressed and the symptoms were controlled. Hyperparathyroidism, malignancy and long-lasting immobilization are the most common causes of hypercalcemia in the elderly. Neuropsychiatric symptoms due to hypercalcemia may occur initially with concentration and increase in sleep time. As the degree of hypercalcemia increases, depression, delirium, confusion and afterwards coma may develop. As described in our patient, acute onset of attention reduction and cognitive or sensory disturbances in the course of delirium are the main features of the fluctuating course. The cause of delirium in elderly patients is multifactorial, so pathologies that may cause delirium should be excluded individually. Delirium is multifactorial, causing serious problems for affected patients, family members, caregivers and healthcare providers. The general purpose of delirium treatment is therefore early diagnosis and treatment with a multidisciplinary approach, which are of great importance in appropriate cases with delirium.

Keywords: Aging, delirium, elderly, hypercalcemia, parathyroid adenoma

Introduction

Delirium can be described as a temporary and reversible brain dysfunction syndrome, which manifests itself primarily with physical, physiopathological and psychological disorders (1). In addition to cholinergic and dopaminergic system disorders, impaired serotonergic system, increased glutamine and glutamate, beta-endorphin reduction, and histaminergic system disruption are thought to be effective in the pathogenesis of delirium (2). Depending on the changes in the concentration of these neurotransmitters in the brain, disturbances of consciousness ranging from mild consciousness to coma may occur. The leading causes of delirium are pneumonia, cancer, urinary infection, electrolyte imbalance (hypo-hyponatremia, hypo-hypercalcemia, hypo-hypermagnesemia), dehydration, congestive heart failure, uremia and stroke. We present a

patient with primary hyperparathyroidism who developed hypercalcemic delirium.

Case Report

A 68-year-old male patient was admitted to the geriatric outpatient clinic with complaints of fatigue, abdominal pain, loss of appetite and weight loss that started about 4 weeks ago. According to the history taken from the relatives of the patient, common pain, delusional thoughts, visual hallucinations, intermittent confusion and agitation attacks were also described. The patient's medical history included hypertension and hyperlipidemia. He had no family history of hypercalcemia or metabolic disease.

In the physical examination, it was observed that he was conscious, awake, cooperative, but not fully oriented in time.

Address for Correspondence: Funda Datlı Yakarıılmaz, Malatya İnönü University Faculty of Medicine, Department of Internal Medicine, Malatya, Turkey

E-mail: fundadatli@gmail.com **ORCID:** orcid.org/0000-0001-5633-0939

Received: 26.11.2021 **Accepted:** 30.03.2023

Cite this article as: Datlı Yakarıılmaz F, Öztürk ZA. Delirium Caused by Hypercalcemia in Older Adults. Eur J Geriatr Gerontol 2023;5(3):262-264



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The total score of the memorial delirium assessment scale (MDAS) used for delirium diagnosis was 13 (>11/30 compatible with delirium) (3). At the end of the evaluation the patient was careless and deterioration was detected in the recall test of three items. His pulse was 96 beats/min and rhythmic, blood pressure was 100/60 mmHg, while respiratory system and abdominal examination were unremarkable. The patient had mild weakness and decreased reflexes in all four extremities, and there was no focal neurologic deficit. The laboratory results of the patient are shown in Table 1.

In order to control the agitation and hallucinations occurring in the patient, after admission to the hospital, haloperidol was given intravenously (iv), 1 mg every 6 hours. In the treatment of hypercalcemia, saline (200 mL/hr iv), zoledronic acid (4 mg iv, once) and dexamethasone (60 mg/day iv) were used. During the first 48 hours of the treatment, the patient's serum calcium level decreased and the symptoms were controlled. MDAS score decreased to 0/30 and hallucinations and agitation findings disappeared. In the following seven days, the patient's mental state continued to improve. Visual hallucinations or agitation were not observed again after the serum calcium level was controlled.

Tests for the etiology of hypercalcemia were performed. No pathological lesion compatible with parathyroid adenoma was observed in the neck ultrasonography. Mediastinal SPECT showed focally increased MIBI involvement in the left upper paratracheal area, which was thought to be an ectopic parathyroid adenoma. Thoracic computed tomography revealed an ectopic parathyroid adenoma.

Discussion

Hypercalcemia is defined as a level of plasma calcium more than 1 mg/dL above the laboratory reference range (4). According to serum calcium levels, 10.5-12 mg/dL is defined as mild, 12-15 mg/dL as moderate, and >15 mg/dL as severe hypercalcemia

	Pre-op	Post-op
Creatinine (0.57-1.25 mg/dL)	1.4	1.1
Sodium (136-145 mmol/L)	131	141
Potassium (3.5-5.1 mmol/L)	3.1	4.2
Magnesium (1.6-2.6 mg/dL)	1.53	1.9
Phosphorus (2.7-4.3 mg/dL)	2.5	3.1
Albumin (3.5-5.0 g/dL)	3.9	4.1
Calcium (8.4-10.2 mg/dL)	15.9	9.8
PTH (10-55 pg/mL)	278	50
25-hydroxyvitamin D (20-70 ng/mL)	30.38	37.5
WBC (4.3-10.3/uL)	10.8	9.7

PTH: Parathyroid hormone, WBC: White blood cell

(hypercalcemic crisis). The most common cause of hypercalcemia is primary hyperparathyroidism and its prevalence in adults ranges from 1-7/1.000 (5). Hyperparathyroidism, malignancy and long-lasting immobilization are the most common causes of hypercalcemia in the elderly (6). When plasma calcium levels are between 10.5-12.0 mg/dL, more than half of the cases are asymptomatic. When calcium levels exceed 13 mg/dL, symptoms and findings that negatively affect many systems are seen (7). Neuropsychiatric symptoms such as impaired concentration and increased sleep duration may occur initially due to hypercalcemia. As the calcium level increases, depression, delirium, confusion, and then coma may develop. As described in our patient, acute onset of attention deficit and cognitive or sensory impairments are the main features of the fluctuating course of delirium. The cause of delirium in elderly patients is multifactorial, so pathologies that may cause delirium should be excluded individually. In our patient, we think that delirium development was caused by hypercalcemia and dehydration. Delirium disappeared after hydration and serum calcium levels were restored to within normal limits. Although hypercalcemia due to overproduction of PTH has been reported with published cases that cause delirium, the pathology of hypercalcemia and delirium has not been clearly established (8). However, calcium is bound to negatively charged membrane proteins, stimulating voltage-dependent sodium channels and decreasing intracellular sodium input. Delirium is multifactorial, and causes serious problems for affected patients, family members, caregivers and healthcare providers. The general purpose of delirium treatment is therefore early diagnosis and treatment with a multidisciplinary approach, which are of great importance in appropriate cases with delirium.

Ethics

Informed Consent: Informed consent was obtained.

Peer-review: Externally peer-reviewed.

Authorship Contributions

Surgical and Medical Practices: F.D.Y., Z.A.Ö., Concept: F.D.Y., Z.A.Ö., Design: Z.A.Ö., Data Collection or Processing: F.D.Y., Analysis or Interpretation: F.D.Y., Literature Search: F.D.Y., Z.A.Ö., Writing: F.D.Y., Z.A.Ö.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

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