

An Unusual Cause of Severe Hypercalcaemia in an Elderly Man

Rianne Parmar*, Tristan Sawle, Daniel Mosby and Ahmed Abdelhafiz

Department of Geriatric Medicine, Rotherham General Hospital, Rotherham, UK

*Corresponding Author: Rianne Parmar, Department of Geriatric Medicine, Rotherham General Hospital, Rotherham, UK.

Received: September 27, 2018; Published: October 31, 2018

Abstract

This case report presents a patient with an unusual cause of severe hypercalcaemia. With alternative causes ruled out, and response to rehydration with intravenous fluid resuscitation, the cause in this case was identified as dehydration. There is only one previously documented case of severe hypercalcaemia caused by dehydration, but with this report in addition, it is important to consider dehydration alone as a cause, and to ensure fully treated before further investigation such as exposure to imaging is carried out.

Keywords: Hypercalcaemia; Dehydration; Acute Kidney Injury; Elderly

Introduction

Hypercalcaemia is defined as adjusted serum calcium level of 2.65 mmol/L or above, on two occasions [1]. It can be classified according to the severity of the adjusted serum calcium level as:

1. Mild: 2.65 - 3mmol/L
2. Moderate: 3.01 - 3.40 mmol/L
3. Severe: > 3.40 mmol/L

Hypercalcaemia is a common disorder, accounting for up to 0.6% of acute hospital admissions [2]. Hypercalcaemia is usually non-symptomatic and often detected by routine blood screening. Symptoms of hypercalcaemia are multi systemic. For example, gastrointestinal manifestations include abdominal pain, nausea, vomiting and constipation; musculoskeletal pain may be noted. Neurologically there may be new onset confusion, depression or psychosis. Renal symptoms such as polyuria, polydipsia and renal colic are often seen. Within the cardiovascular system there may be hypertension, arrhythmias and bradycardia. Mild hypercalcaemia is not normally symptomatic [3]. Most patients show symptoms at moderate to high levels of calcium only. Severity of symptoms is likely to be correlated with the severity of hypercalcaemia. Hypercalcaemia is most commonly due to primary hyperparathyroidism, followed closely by malignancy [4]. A full list of causes can be found in table 1. Management of hypercalcaemia generally involves correcting dehydration with intravenous fluids, promotion of diuresis with intravenous diuretics and the use of bisphosphonates if hypercalcaemia persists. Specific measures will be required, depending on the aetiology of the hypercalcaemia, for example, parathyroidectomy for hyperparathyroidism or treatment of malignancy [5].

1. Malignancy
2. Primary hyperparathyroidism
3. Tertiary hyperparathyroidism
4. Vitamin D intoxication
5. Medications (e.g. thiazides and lithium)
6. Endocrine disorders (e.g. hyperthyroidism, acromegaly)
7. Genetic disorders (e.g. familial hypocalciuric hypercalcaemia)
8. Dehydration

Table 1: Causes of hypercalcaemia.

Case Report

Present history: An 83 year old man presented with a two week history of increased confusion. His family noted mumbling speech and he himself had noticed that his mobility had reduced over this period of time, despite the use of a walking aid. He denied any headache, fever, shortness of breath, pain or visual symptoms.

Examination: Vital signs were all normal. Physical examination was unremarkable other than dry mucous membranes, a GCS of 14 (due to confusion) and an AMT score of 6/10. He had a mild scattered wheeze and the abdomen was soft with no organomegaly. He was able to move all limbs normally and had no cerebellar signs.

Medications on admission: Amitriptyline, candesartan, adcal D3 (Calcium carbonate (750 mg) and vitamin D3 (200 I.U), nebivolol, indapamide, codydramol and amlodipine.

Past medical history: Bilateral knee replacement, rheumatoid arthritis, hypertension, mild chronic kidney disease (CKD).

Investigations: The following investigations were undertaken:

- Inflammatory markers, haemoglobin and liver function tests (LFTs) were normal.
- Sodium and potassium were within normal ranges but the urea was raised at 14.19 mmol/L and creatinine at 186 µmol/L, showing an acute kidney injury (AKI).
- The adjusted calcium was found to be raised at 3.63 mmol/L. Parathyroid hormone (PTH) was reduced, Vitamin D was normal.
- CT head showed no acute changes.
- Chest radiography was normal.
- Myeloma screen and urinary Bence-Jones protein were negative
- ECG showed sinus rhythm, with T wave depression in leads V3-4.

Management: IV fluid infusions were given, co-dydramol, candesartan, adcal D3 were held due to acute kidney injury. IV fluids were continued, furosemide was added as well as pamidronate infusion. At this point in time, around two weeks into admission, he was still disoriented in time, place and person and actively confabulating. Due to the unknown cause of hypercalcaemia, malignancy was suspected and the patient underwent the relevant investigations such as CT chest/abdomen/pelvis which showed no signs suggestive of malignancy. A bone isotope scan of the whole body was negative. A few days later, as adjusted calcium level was down to 2.84 mmol/L, patient became less confused. At some point, we felt that he was overloaded and intravenous fluid was withheld temporarily which led to another surge of calcium level to a peak of 3.3 mmol/L. However, he remained stable again after re-starting fluid infusion for the rest of his stay in hospital

for almost 4 weeks and serum calcium was 2.19 mmol/L on discharge. On outpatient clinic review, he remained very well, AMT was 9/10 and he maintained his serum calcium within normal ranges.

Discussion

We have presented an 83 year old man with dehydration and reversible hypercalcaemia, corrected by simple rehydration measures. His main presentation was confusion, reflecting the dominance of neuropsychiatric symptoms in this age group. Dehydration is an unusual cause of severe hypercalcaemia not commonly documented in the literature. Only one case was reported in the literature of severe hypercalcaemia secondary to dehydration in a younger woman 63 years of age [6]. Her dehydration was due to vomiting and resulted in toxic megacolon and an urticarial rash. This was managed with aggressive rehydration and no other cause was found. In our case, there was no history of fluid loss but the patient’s AKI suggested that he was dehydrated. As his renal function improved (and therefore his level of hydration), the adjusted calcium level normalised (Figure 1). Dehydration is common in older people, often due to poor oral intake. Hypercalcaemia in older people may begin mild and asymptomatic but if allowed to progress and cause mental dysfunction, it could lead to further cognitive impairment and severe dehydration. This can end up a vicious cycle, resulting in acute kidney injury and further dehydration and confusion. As confusion was the initial presentation, an AMT was done and monitored frequently throughout the patient’s stay in hospital to measure progress. Calcium is commonly measured in a ‘delirium screen’. As our patient’s creatinine and adjusted calcium levels normalised, so did his AMT score, showing a steady improvement in symptoms. This gentleman was taking vitamin D supplementation, but the cause of hypercalcaemia was unlikely to be vitamin D intoxication due to his vitamin D levels being within the normal

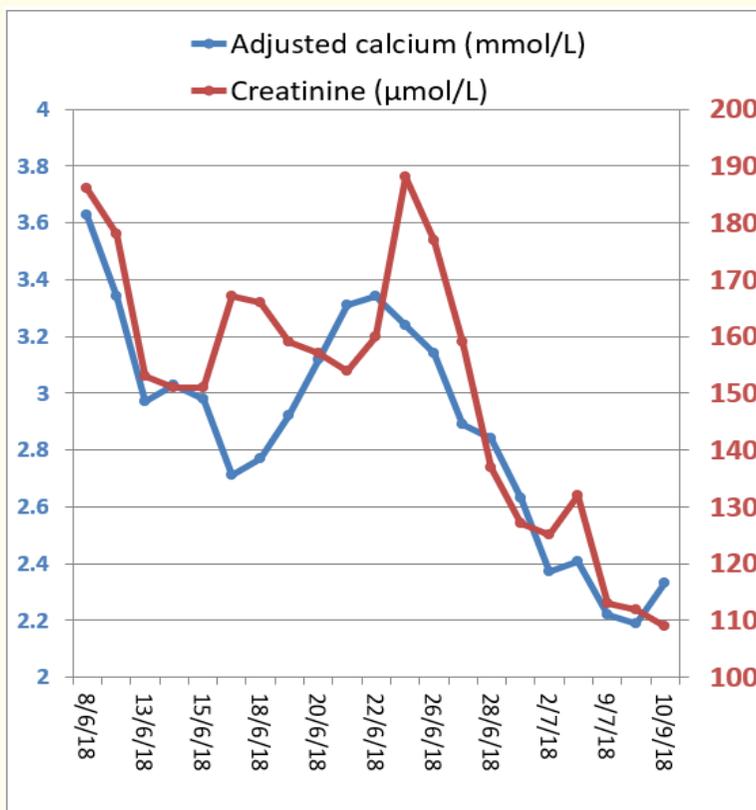


Figure 1: Graph of patient’s adjusted calcium and creatinine levels over time.

range. Our patient was also taking indapamide modified release at a dose of 1.5mg per day, a “thiazide-like” diuretic. His hypercalcaemia was corrected with rehydration despite continuation of the drug at the same dose, pointing towards that dehydration as the likely cause. Thiazide associated hypercalcaemia is poorly understood, often associated with primary hyperparathyroidism (demonstrably not the case in our patient) and does not appear to present as severe hypercalcaemia, often only presenting with mild hypercalcaemia, therefore we believe this is unlikely to be the culprit in our case [7,8]. In the absence of other cause and his rapid response to rehydration indicates that dehydration was likely to be the cause of both hypercalcaemia and acute kidney injury. Confusion and other neuropsychiatric symptoms are likely caused by the depressant effects of high serum calcium. This is easily and fully reversible with fluids. However, patients presenting with hypercalcaemia and confusion often undergo imaging quite early to rule out malignancy. This exposes them to radiation before a much simpler cause of their symptoms is completely ruled out. It is worth considering completion of rehydration to see if calcium is corrected before putting patient under unnecessary exposure.

Learning Points

- Hypercalcaemia is common in older people.
- Confusion is the commonest clinical manifestation of hypercalcaemia in older people.
- The correction of dehydration and normalisation of calcium is essential before exposing patients to unnecessary investigations to avoid high doses of radiation.

Conclusions

Dehydration-related hypercalcaemia in older people is less reported in the literature. Dehydration is commonly associated with mild hypercalcaemia. However, older people may be vulnerable to the progression of mild to severe dehydration-related hypercalcaemia due to the negative effect of hypercalcaemia on cognitive function which further impairs thirst sensation that leads to further worsening of dehydration and the severity of hypercalcaemia. Therefore, simple rehydration measures should be undertaken before embarking on unnecessary extensive radiological investigations.

Bibliography

1. Smellie WS, *et al.* “Best practice in primary care pathology: review 11”. *Journal of Clinical Pathology* 61.4 (2008): 410-418.
2. Dent DM, *et al.* “The incidence and causes of hypercalcaemia”. *Postgraduate Medical Journal* 63.743 (1987): 745-750.
3. Carroll M and Schade D. “A Practical Approach to hypercalcaemia”. *American Family Physician* 67.9 (2003): 1959-1966.
4. Turner JJO. “Hypercalcaemia - presentation and management”. *Clinical Medicine* 17.3 (2017): 270-273.
5. Longmore M, *et al.* “The Oxford Handbook of Clinical Medicine”. Oxford University Press, 9th Edition (2014): 690-691.
6. Fernandes LG, *et al.* “Severe hypercalcaemia and colon ischaemia: dehydration as an unusual cause?” *BMJ Case Reports* (2015): bcr2014208809.
7. Carrol R and Matfin G. “Endocrine and metabolic emergencies: hypercalcaemia”. *Therapeutic Advances in Endocrinology and Metabolism* 1.5 (2010): 225-234.
8. Griebeler ML, *et al.* “Thiazide-Associated Hypercalcemia: Incidence and Association With Primary Hyperparathyroidism Over Two Decades”. *Journal of Clinical Endocrinology and Metabolism* 101.3 (2016): 1166-1173.

Volume 3 Issue 6 November 2018

© All rights reserved by Rianne Parmar, *et al.*