**Is there more than one culprit?**

**An interesting case of multi-factorial hypercalcaemia**

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**Introduction**

Hypercalcaemia can be prevalent in up to 3% of the population.\(^1\)

Calcium levels are tightly regulated through parathyroid function, bone resorption, renal calcium reabsorption and dihydroxylation of vitamin D. Any failure in these mechanisms will lead to elevated calcium levels.\(^2\)

This case highlights the importance of considering multiple aetiologies in patients presenting with hypercalcaemia.

**Case Report**

**Initial Presentation**

A 40-year-old Asian gentleman with a background of bipolar disorder and type 2 diabetes was referred to the acute medical team after routine blood tests demonstrated acute hypercalcaemia. It was noted that the patient was on lithium and cholecalciferol at the time.

Further investigations revealed that he was lithium toxic. Both medications were ceased, the patient was rehydrated and subsequently discharged.

**Second admission:**

He re-presented 6 weeks later with severe hypercalcaemia (corrected calcium of 3.41 mmol/L) and acute renal failure with a creatinine of 232. This was despite being off the initial offending medications.

Tests performed on this admission revealed low parathyroid hormone levels, low vitamin D levels and a significantly raised serum angiotensin-converting enzyme levels of 101 u/L.

High resolution CT demonstrated widespread paratracheal, supracarinal and hilar lymphadenopathy, raising the possibility of underlying sarcoidosis. He subsequently had an axillary node biopsy which showed non-caseating granulomas.

Following a 2 week course of oral prednisolone, the patient’s calcium levels normalised and the renal function significantly improved.

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**Imaging**

![Figure 1: Admission chest film showing a degree of hilar lymphadenopathy](image1)

![Figure 2: CT Chest showing several areas of lymphadenopathy (axillary and mediastinal).](image2)

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**Discussion**

In up to 90% of cases, hypercalcaemia is a result of either primary hyperparathyroidism or malignancy.\(^3\) Other causes include drugs such as lithium, granulomatous diseases such as sarcoidosis and chronic kidney disease.\(^2\) This case highlights the importance of considering more than one cause in patients presenting with hypercalcaemia and raises the awareness of ensuring such patients are fully investigated with appropriate tests.

**Investigating Hypercalcaemia**

<table>
<thead>
<tr>
<th>Hypercalcaemia</th>
<th>History</th>
<th>Examination</th>
<th>Investigations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild (&lt;3.0 mmol/L)</td>
<td>• Symptoms / duration • Underlying systemic disease • Family history • Full drug history</td>
<td>• Assess cognition • Assess for lymphadenopathy • Breast examination • Fluid balance status</td>
<td></td>
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<tr>
<td>Moderate (3.0-3.5 mmol/L)</td>
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<td></td>
<td>• Magnesium and phosphate levels • Urea and electrolytes • Vitamin D levels and Parathyroid hormone</td>
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<tr>
<td>Severe (&gt;3.5 mmol/L)</td>
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If malignancy screen negative

- Consider other causes
  1. Sarcoidosis (serum angiotensin converting enzyme).
  2. Thyrotoxicosis (Thyroid function tests).
  3. Immobilisation.
  4. Adrenal insufficiency (serum cortisol).
  5. Phaeochromocytoma.
  6. Familial hypocalcic hypercalcaemia (urine calcium excretion).

**References**


Lewisham and Greenwich NHS