

Severe Hypercalcaemia and Pancreatitis in a 14-Year-Old with Generalised Lymphadenopathy: A Diagnostic Challenge Between Lymphoma and Tuberculosis

JaC Sameer Atala Abdo1*, Khaled Siddiqui2, Mohamed Hammad3 and Jisna Palliyal3

¹Clinical Fellow, Department of General Paediatrics, East Lancashire Hospitals NHS Trust, Blackburn, UK

*Corresponding Author: JaC Sameer Atala Abdo, Department of General Paediatrics, East Lancashire Hospitals NHS Trust, Blackburn, UK.

Received: November 10, 2025; Published: November 20, 2025

Abstract

Background: Hypercalcaemia is an uncommon finding in children and most often raises suspicion of malignancy. Granulomatous diseases such as tuberculosis (TB) are rare but important alternative causes.

Case Presentation: A 14-year-old boy was referred by his GP for concerns of abdominal pains and vomiting, with weight loss, and anorexia. The child was seen in Paediatric assessment Unit, Examination revealed Pallor, dehydration, hypertension, epigastric tenderness. Investigations done and revealed severe hypercalcaemia, pancreatitis, suppressed parathyroid hormone, and widespread lymphadenopathy. Lymphoma was initially suspected. However, lymph node biopsy demonstrated granulomatous inflammation, a Mantoux test was strongly positive, and there was a history of TB exposure. He was commenced on anti-tuberculous therapy with resolution of symptoms and normalisation of calcium.

Conclusion: Disseminated TB can mimic haematological malignancy in children. Awareness of this presentation is essential, particularly in patients with TB risk factors, to avoid misdiagnosis and unnecessary chemotherapy.

Keywords: Hypercalcaemia; Tuberculosis; Paediatrics; Pancreatitis; Lymphadenopathy; Case Report

Introduction

Hypercalcaemia in childhood is rare, and when present, most commonly reflects malignancy, primary hyperparathyroidism, or granulomatous disease [1,2]. Tuberculosis remains a major global health challenge, and extrapulmonary disease can manifest with lymphadenopathy, systemic symptoms, and metabolic complications [3,4].

The pathophysiology of hypercalcaemia in granulomatous disease is attributed to extrarenal production of calcitriol by activated macrophages [5]. Clinical differentiation from lymphoma can be extremely difficult, requiring tissue confirmation.

We report a case of disseminated TB presenting with severe hypercalcaemia and pancreatitis in an adolescent boy, highlighting the diagnostic overlap with malignancy.

²Senior Paediatrician, East Lancashire Hospitals NHS Trust, Blackburn, UK

³SCF Paediatrics, East Lancashire Hospitals NHS Trust, Blackburn, UK

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Case Presentation

Patient information

A 14-year-old boy presented with a 3-month history of progressive weight loss and a 1-month history of epigastric pain, vomiting, and anorexia. There was no history of night sweats or haemoptysis. His past medical history was unremarkable. His grandmother had previously been treated for TB, and he had recently travelled to Pakistan.

Clinical findings

On admission, he was pale, dehydrated, and hypertensive. Abdominal examination revealed epigastric tenderness. No palpable superficial lymph nodes were noted. Ophthalmology assessment ruled out ocular TB.

Diagnostic assessment

Laboratory results:

- Serum calcium: 4.39 mmol/L (severe hypercalcaemia)
- Amylase: 538 U/L (acute pancreatitis)
- PTH: Suppressed
- · Uric acid and LDH: Elevated
- Thyroid function: Transient hyperthyroidism, later normalised
- Mantoux: 15 mm induration
- MRSA CPE screens negative
- HTLV PCR neg
- HIV/Hep A, B, E screen negative
- CMV negative
- Measles IgG negative
- VZV IgG positive.

Imaging

CT scan revealed widespread mediastinal and abdominal lymphadenopathy, splenomegaly, and pulmonary consolidation.

Histopathology

Excisional lymph node biopsy showed granulomatous inflammation with no evidence of malignancy.

Differential diagnosis

- Lymphoma: Supported by weight loss, lymphadenopathy, raised urate and LDH.
- Tuberculosis: Supported by granulomatous biopsy, positive Mantoux, family exposure.
- Sarcoidosis: Less likely in paediatric age group but considered.

Timeline

Time (relative)	Event
-3 months	Progressive weight loss begins
-1 month	Onset of abdominal pain, vomiting, anorexia
Admission (Day 0)	Found pale, dehydrated, hypertensive; investigations show severe hypercalcaemia, pancreatitis
Day 2	Imaging shows widespread lymphadenopathy and splenomegaly
Day 5	Lymph node biopsy performed
Day 10	Biopsy shows granulomatous inflammation; Mantoux positive
Week 2	Commenced on anti-tuberculous therapy
3 months follow-up	Clinical and biochemical improvement; calcium normalised

Table 1

Discussion

This case illustrates the diagnostic challenge of differentiating TB from malignancy in children presenting with hypercalcaemia and generalised lymphadenopathy.

Mechanism of hypercalcaemia in TB: Activated macrophages in granulomatous inflammation produce 1α -hydroxylase, converting 25-hydroxyvitamin D to calcitriol, leading to increased calcium absorption [6].

Pancreatitis: Severe hypercalcaemia can trigger acute pancreatitis, as seen in our patient [7,8].

Differential diagnosis: The combination of weight loss, lymphadenopathy, and elevated LDH initially suggested lymphoma. However, histopathological findings and TB exposure risk were decisive in guiding diagnosis.

Teaching point: Clinicians should maintain a broad differential when evaluating paediatric hypercalcaemia and avoid anchoring on malignancy alone. Awareness of TB as a rare but important cause prevents unnecessary oncological interventions.

Conclusion

Disseminated TB should be considered in children presenting with severe hypercalcaemia and widespread lymphadenopathy, particularly in those with epidemiological risk factors. Tissue biopsy is critical in distinguishing TB from malignancy. Early recognition facilitates timely treatment and avoids inappropriate chemotherapy.

Learning Points

- Tuberculosis may present with widespread lymphadenopathy and severe hypercalcaemia in children.
- Travel and family history are essential in evaluating suspected malignancy.
- Granulomatous disease should always be considered in non-PTH-mediated hypercalcaemia.
- A multidisciplinary approach is essential for diagnosis and management.

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