**Sri Lanka Journal of Medicine** **SLJM**

**Case Report**

Citation Ekanayake TD, Rathnayake RMPM, Dissanayake AMSDM et al. 2026 Bone Marrow Fibrosis as a Rare Manifestation of Primary Hyperparathyroidism Due to Parathyroid Adenoma. Sri Lanka Journal of Medicine, pp. 68-70.
DOI: https://doi.org/10.4038/sljm.v35i1.650

# Bone Marrow Fibrosis as a Rare Manifestation of Primary Hyperparathyroidism Due to Parathyroid Adenoma

*TD Ekanayake[1], RMPM Rathnayake[1], AMSDM Dissanayake[1], GCM Weerasooriya[1]*

[1]Teaching Hospital, Peradeniya, Sri Lanka

**Correspondence:**
TD Ekanyake
E mail: thilinide17@gmail.com
https://orcid.org/0009-0000-6956-1381

## ABSTRACT

Primary hyperparathyroidism (PHPT) typically presents with hypercalcaemia and skeletal changes but rarely causes haematological manifestations. We present a case report of a 66-year-old woman presented with refractory pancytopenia and bone marrow fibrosis. She was found to have hypercalcaemia and elevated parathyroid hormone (PTH), with a parathyroid adenoma. She was started on vitamin D supplementation and referred for surgical resection of the adenoma.

*Keywords: Hyperparathyroidism, pancytopenia, bone marrow fibrosis*

## INTRODUCTION

Primary hyperparathyroidism (PHPT) is most commonly caused by a parathyroid adenoma and classically presents with hypercalcaemia, renal calculi, neuropsychiatric disturbances, and skeletal manifestations [1]. Less commonly, PHPT can lead to haematological abnormalities such as anaemia or pancytopenia due to secondary bone marrow fibrosis [2]. We report a rare case where PHPT was the underlying cause of bone marrow fibrosis in a patient with unexplained cytopenias.

## CASE REPORT

A 66-year-old woman presented with progressive fatigue and weight loss over two months. She denied bleeding, recurrent infections, or constitutional symptoms such as night sweats or fever. Clinical examination revealed pallor and mild icterus, but no lymphadenopathy, hepatosplenomegaly, or bone tenderness.

Initial investigations revealed pancytopenia: haemoglobin 4.1 g/dL, white cell count 2.97 × $10^9$/L, and platelet count 107 × $10^9$/L. Reticulocyte count was low. There was no evidence of haemolysis. Liver and renal function tests were within normal limits. Peripheral blood smear showed normocytic normochromic anemia with macrocytes and tear drop cells without blasts or dysplastic features.

Bone marrow aspiration yielded a dry tap. Trephine biopsy showed thickened bony trabecule architecture with markedly suppressed trilineage





**Received: 2025-09-26** **Accepted revised version: 2026-04-07** **Published: 2026-04-22**

haematopoiesis with streaming of fibroblast proliferation as shown in Figure 1.

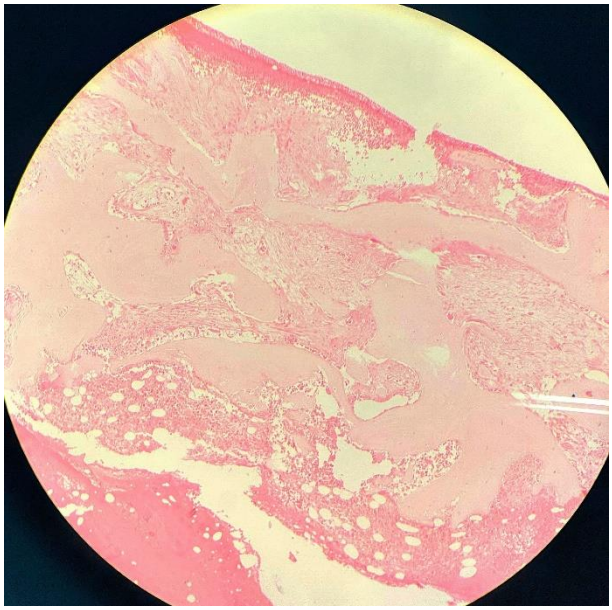

**Figure 1: Bone marrow trephine biopsy - H & E staining showing thickened bony trabecular architecture with suppressed trilineage haematopoiesis and increased fibroblast proliferation**

Reticulin stain showed grade 3 fibrosis as shown in Figure 2. Immunohistochemistry did not reveal evidence of haematological or metastatic malignancy. MPN panel and BCR-ABL mutations were negative.

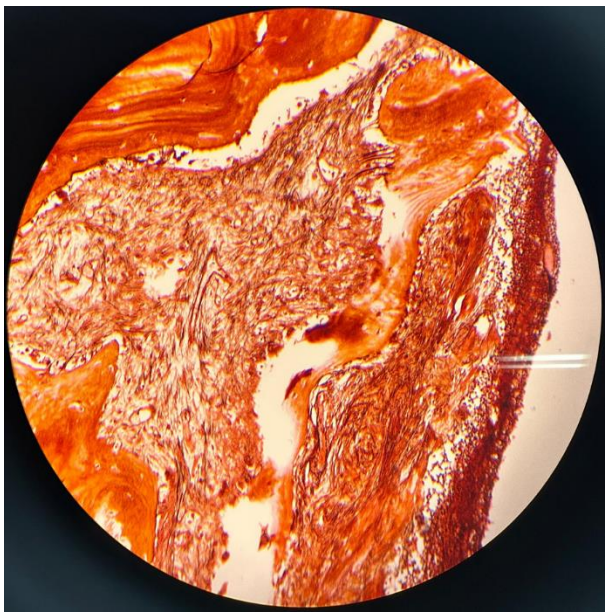

**Figure 2: Bone marrow trephine biopsy reticulin stain showing grade 3 fibrosis.**

Further evaluation revealed hypercalcaemia (corrected calcium 2.95 mmol/L), marginally low phosphate, and elevated intact parathyroid hormone (PTH) levels of 2430 pg/mL. Vitamin D levels were low. Dual-energy X-ray absorptiometry (DEXA) scan showed osteoporosis. Neck ultrasound identified a hypoechoic lesion posterior to the left thyroid lobe, consistent with a parathyroid adenoma.

She was started on vitamin D supplementation to correct deficiency and stabilize calcium levels pre-operatively. She was referred for parathyroidectomy to remove the adenoma. Later surgical removal of parathyroid adenoma was performed and histology also confirmed it as parathyroid adenoma. Following surgery her pancytopenia was improved and within two months full blood count parameters were normalized. Hypercalcaemia also normalized.

## DISCUSSION

Bone marrow fibrosis is an uncommon but potentially reversible complication of PHPT [2]. High levels of PTH are thought to promote fibroblast proliferation and cytokine release, resulting in marrow fibrosis and pancytopenia [3]. This rare manifestation can mimic myeloproliferative disorders or marrow infiltration by malignancy [4].

Previous reports have demonstrated hematologic improvement and regression of fibrosis following successful parathyroidectomy [5,6]. Early recognition and treatment are essential to avoid unnecessary hematologic workups or therapies. This case highlights the importance of considering endocrine aetiologies in unexplained pancytopenia. Major learning points in this case report include, PHPT may rarely cause secondary bone marrow fibrosis and pancytopenia, Excess PTH can stimulate fibroblast activity in the marrow. This condition is potentially reversible with parathyroidectomy and endocrine evaluation is crucial in unexplained cytopenias with marrow fibrosis.

**Author declaration**

**Acknowledgement**
Authors thank all the staff engaged in diagnosis and management of this patient members of this patient who consented for this publication.

**Authors' contributions:**
TDE: Conceptualization, design, data collection, patient management, manuscript writing; GCMW: Data collection, patient management; RMPMR: Conceptualization, design, patient management, manuscript writing; DMD: Conceptualization, design Patient management.

**Conflicts of interest:**
The authors declare that there is no financial or non-financial conflict of interest.

**Funding statement:**
Self-funded.

**Ethics statement:**
Written informed consent was obtained from the patient before they participated in the case, ensuring their understanding of the purpose, procedures, and potential implications involved.

**Statement on data availability:**
All data are available upon request.

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