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Images in Nephrology

Hyperparathyroid bone disease

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A 52-year-old haemodialysis patient developed normochromic normocytic anaemia requiring weekly blood transfusions. He was resistant to high-dose epoetin alfa and intravenous iron therapy. A blood film examination revealed tear-drop poikilocytes. Serum B12, red cell folate, iron studies, serum protein electrophoresis, serum-free light-chain assay and haemolysis screen were all normal. The corrected serum calcium level was 2.43 mmol/L (2.15–2.55), phosphate 1.58 mmol/L (0.81–1.45) and PTH 225 pmol/L (1.0–7.0), consistent with tertiary hyperparathyroidism attributable to end-stage renal disease.

Bone marrow examination revealed a dry tap. Bone marrow trephine was grossly abnormal. The bone trabeculae demonstrated marked paratrebecular fibrosis as seen in Figure 1, and prominent Howship's Lacunae (containing osteoclasts and forming pits in the bone surface as marked in Figure 2). There were large numbers of multinucleate foreign body-type giant cells consistent with severe hyperparathyroid bone disease (osteitis fibrosa cystica) as marked in Figure 3.

The patient underwent subtotal parathyroidectomy, which was followed by a rapid improvement in his anaemia and

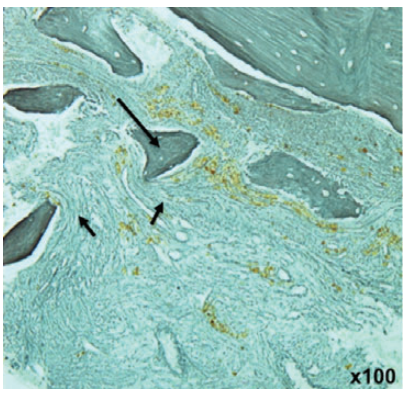

Fig. 1. Reticulin stain demonstrating trabeculae (long arrow) with marked para-trabecular fibrosis (short arrows).

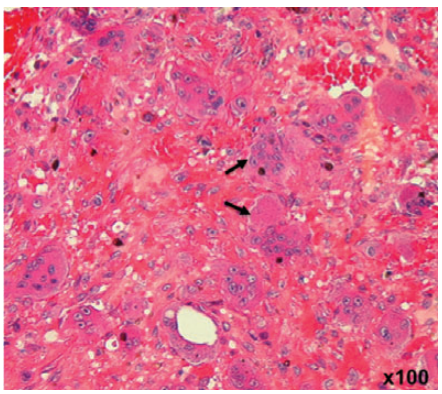

Fig. 3. H&E stain showing infiltration of multi-nucleate foreign body-type giant cells (arrows).

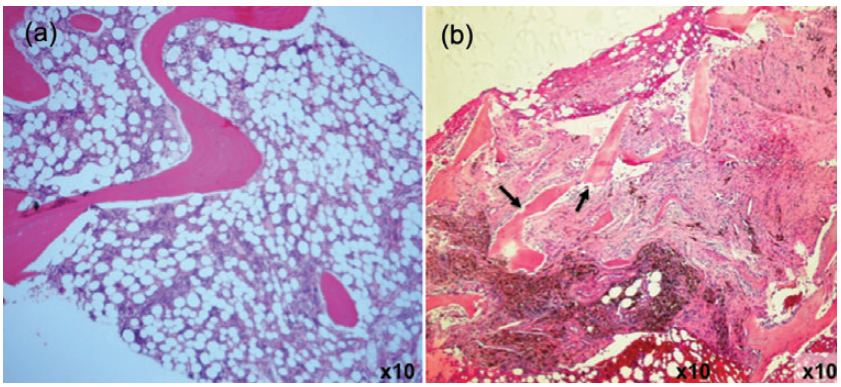

Fig. 2. H&E stain (**a**) normal and (**b**) demonstrating hypercellularity, multi-nucleate giant cells and prominent Howship's Lacunae (arrows).

subsequent reduction of the dose of epoetin alfa. A bone marrow examination after 9 months showed a reduction in the number of giant cells and a modest reduction in marrow fibrosis.

The osseous effects of excess parathyroid harmone (PTH), which include marrow fibrosis, are paramount in the aetiology of anaemia in osteitis fibrosa cystica, but the overall mechanism remains incompletely understood. It is likely to involve interaction between bone marrow stroma, cytokines and mast cells. Other postulated effects include inhibition of endogenous erythropoietin synthesis, haeme synthesis and impaired red cell survival. The extent of marrow fibrosis seems to correlate well with the severity of anaemia and the required dose of exogenous erythropoiesis stimulating agents (ESAs) [1, 2].

In the case of unexplained resistance to exogenous ESAs, investigation of secondary hyperparathyroidism is recommended, and where needed, a bone biopsy should be performed. Both anaemia and marrow fibrosis improve after curative parathyroidectomy with reduction in the required dose of ESAs.

Conflict of interest statement. None declared.

References

1. Rao DS, Shih MS, Mohini R. Effect of serum parathyroid hormone and bone marrow fibrosis on the response to erythropoietin in uremia. *N Engl J Med* 1993; 328: 171–175
2. Urena P, Eckardt KU, Sarfati E *et al*. Serum erythropoietin and erythropoiesis in primary and secondary hyperparathyroidism: effect of parathyroidectomy. *Nephron* 1991; 59: 384–393

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