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Back Pain and Lower Extremity Sensory Loss in an ESKD Patient

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Case Description

A 33-year-old man on maintenance hemodialysis for 6 years for ESKD secondary to IgA nephropathy presented with acute back pain and loss of sensation in his feet, in the absence of recent trauma. His past medical history included severe hyperparathyroidism, with intact parathyroid hormone levels (iPTH) more than ten times the upper limit of the normal range (80 pg/ml) for 4 years, despite the prescription of phosphate binders, calcitriol, cinacalcet, and—lately—etelcalcetide. Clinical examination was marked by symmetric sensory loss and weakness in the lower extremities, together with deep-tendon hyperreflexia and Babinski signs. Plasma levels of electrolytes were normal. iPTH (855 pg/ml) and alkaline phosphatase (500 IU/L; normal range, 40–130 IU/L) levels were unchanged. Magnetic resonance imaging of the spine revealed diffuse bone-marrow infiltration and a fracture of the third thoracic vertebra causing cord compression (Figure 1A, arrow).

The patient underwent urgent spinal decompression, combining laminectomy and osteosynthesis. Surgical exploration revealed a heterogeneous mass with bloody and fleshy components. Blood cultures and microbiologic testing of the surgical sample were negative. Histopathologic examination showed numerous, multinucleated giant cells (Figure 1B, black arrows) with hemosiderin deposits (Figure 1B, white arrow), consistent with a brown tumor. Total-body bone scintigraphy showed disseminated increased uptake involving the axial skeleton. The patient fully recovered from paraparesis a few weeks after surgery. He received a deceased-donor kidney transplant 8 months later, after which iPTH levels returned to normal.

Discussion

A brown tumor is a focal osteolytic lesion caused by high bone turnover due to uncontrolled and long-standing hyperparathyroidism (1–3). It is composed of collections of osteoclasts intermixed with fibrous tissue and poorly mineralized woven bone. It is characterized by the presence of numerous giant cells and hemosiderin deposits that cause the reddish-brown color (2). It predominantly affects the mandibula, clavicles, long bones, pelvis, and ribs (1–3). Spinal involvement is rare (2). The lesion is seen as single or multiple well-demarcated radiolucent zones (geodes), without invasion of adjacent tissues. A brown tumor is asymptomatic unless there is adjacent structure compression or pathologic fracture (3). It should be distinguished from malignant bone lesions, spondylodiscitis, and spondyloarthropathy due to β2-microglobulin amyloidosis (4). The final diagnosis is made after histopathologic examination, coupled with the clinical and laboratory history of severe hyperparathyroidism (3). Correction of the underlying hyperparathyroidism usually leads to tumor regression (1–3). Resection of the brown tumor is required only in those who do not respond to medical therapy or in patients with severe pain or neurologic deficits (1–3).

Teaching Points

- A brown tumor is a rare complication of severe hyperparathyroidism, which should be considered in patients with ESKD presenting with an osteolytic bone lesion.
- Histologic examination is characterized by the presence of numerous giant cells and hemosiderin deposits.
- The brown tumor is managed by correcting the underlying hyperparathyroidism.

Disclosures

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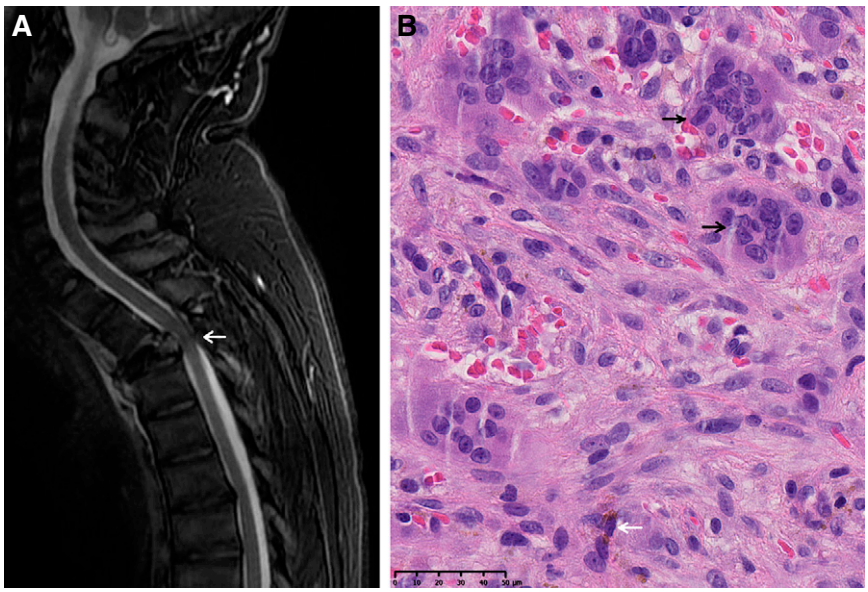

Figure 1. | **Brown tumor of the thoracic spine.** (A) Magnetic resonance imaging of the spine reveals diffuse bone-marrow infiltration and fracture of the third thoracic vertebra, causing cord compression (arrow). (B) Light microscopy shows multiple multinucleated giant cells (black arrows), stromal cells, and hemosiderin deposits (white arrow). Hematoxylin and eosin stain. Original magnification, ×40.

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Author Contributions

L. Labriola reviewed and edited the manuscript; and C. Maalouly and B. Vô wrote the original draft.

References

1. Can Ö, Boynueğri B, Gökçe AM, Özdemir E, Ferhatoğlu F, Canbakan M, Şahin GM, Titiz MI, Apaydın S: Brown tumors: A case report and review of the literature. *Case Rep Nephrol Dial* 6: 46–52, 2016
2. Duval-Sabatier A, Gondouin B, Bouvier C, Bataille S, Berland Y, Brunet P: Brown tumor: Still an old disease? *Kidney Int* 80: 1110, 2011
3. Di Daniele N, Condò S, Ferrannini M, Bertoli M, Rovella V, Di Renzo L, De Lorenzo A: Brown tumour in a patient with secondary hyperparathyroidism resistant to medical therapy: Case report on successful treatment after subtotal parathyroidectomy. *Int J Endocrinol* 2009: 827652, 2009
4. Labriola L, Jadoul M: Dialysis-related amyloidosis: Is it gone or should it be? *Semin Dial* 30: 193–196, 2017

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