Case Description

A 51-year-old man with ESKD secondary to FSGS on thrice weekly hemodialysis presented with an expanding painful right arm mass. He had long-standing secondary hyperparathyroidism with a persistently elevated PTH level > 2000 pg/ml and had been refusing to take calcimimetics or to undergo parathyroidectomy.

Physical examination demonstrated a hard and tender mass on the lateral aspect of the humerus.

An x-ray showed a 7.2×4-cm expanding lytic lesion with periosteal reaction (Figure 1). A skeletal survey showed similar smaller masses in the left humerus, left second metatarsal bone, and distal ulna together with few calcified soft tissue lesions in the right forearm, as well as partial Achilles tendon calcification and calcified abdominal aorta. A diagnosis of brown tumor was made on the basis of his longstanding hyperparathyroidism.

Brown tumors are rare, non-neoplastic, lytic bony lesions that can develop in patients with poorly controlled hyperparathyroidism. They are usually diagnosed between the third and sixth decades of life with a female predominance (1,2). Their prevalence has been reported to be around 3% in primary hyperparathyroidism and varies from 1.5% to 13% in secondary hyperparathyroidism (3).

Brown tumors can be solitary or multifocal. These destructive lesions result from increased bone remodeling secondary to upregulated osteoclastic activity (1). They can affect any skeletal bone but the most common sites are the extremities, pelvis, ribs, clavicles, and mandible. They have also been reported to affect the craniofacial bones, sternum, and spine (3,4).

The clinical presentation of brown tumors varies depending on the lesion size and the affected site. They can be asymptomatic and diagnosed incidentally on imaging. When symptomatic, brown tumors can present as painful bony enlargements, pathologic fractures, or they can result in compressive manifestations such as spinal cord compression symptoms (2,4).

Radiologically, brown tumors have a cyst-like radiolucent appearance on x-rays. When multiple, they can mimic metastatic disease or multiple myeloma. They appear as enhancing hypervascular lesions on contrast computed tomography scans and angiography. On magnetic resonance images, brown tumors exhibit hypointense signals on T1-weighted images, and they can be either hypointense or hyperintense on T2-weighted images (2,3).

Microscopically, brown tumors are composed of highly vascularized fibroblastic stroma, rich in spindle-like stromal cells, fibroblasts, osteoclastic multinucleated giant cells, and hemosiderin-laden macrophages. Microfractures in this vascular regenerative tissue result in bleeding and hemosiderin pigment deposition, giving these brown tumors their pathognomonic color and name. Another pathologic feature of these high-turnover lesions is the formation of peripheral poorly mineralized bone (osteoid) by osteoblasts (1,5).

Control of hyperparathyroidism either medically or surgically by parathyroidectomy represents the first-line therapy for brown tumors. Surgical intervention is
reserved for patients where erosive lesions adversely affect the surrounding organs causing neurologic compromise, cauda equina symptoms, or facial deformity. Surgery has also been reported when the tumor fails to regress within 6 months to 2 years of hormonal control (2–4).

**Teaching Points**

- Brown tumors represent an extreme form of renal osteodystrophy. They develop in ESKD patients with uncontrolled hyperparathyroidism.
- Brown tumors may be asymptomatic or present with painful mass and/or compressive symptoms related to surrounding tissue involvement.
- First-line treatment of brown tumors is control of hyperparathyroidism, either medically or surgically.

**Author Contributions**

D. Gebrael, S. Jdiaa, and S. Koubar wrote the initial draft, reviewed and edited the manuscript, and contributed to patient care.

**Disclosures**

All authors have nothing to disclose.

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**References**


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