Research Article

Suspicion Of Brown Tumor In A Young Woman Undergoing Maintenance Hemodialysis In A Rural Area: A Rare Case Report

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ABSTRACT

Brown tumor is a focal bone lesion caused by increased osteoclastic activity secondary to uncontrolled hyperparathyroidism. Although commonly associated with primary hyperparathyroidism, it rarely occurs in secondary hyperparathyroidism (SHPT) among young patients undergoing long-term hemodialysis. We report a rare and diagnostically challenging case of a 28-year-old woman on maintenance hemodialysis for seven years who presented with dyspnea, productive cough, and a progressively enlarging painless palatal mass causing difficulty in mastication, accompanied by a significant height loss of 13 cm. Laboratory investigations revealed markedly elevated intact parathyroid hormone (iPTH) levels (3,239 pg/mL), increased alkaline phosphatase (1,721 U/L), hypocalcemia (8.4 mg/dL), and vitamin D deficiency (14.4 ng/mL). Panoramic radiography demonstrated a multilocular lytic lesion with expansive involvement of the right maxilla, while fine-needle aspiration biopsy suggested a vascular lesion with abundant erythrocytes. Based on integrated clinical, biochemical, and radiological findings, the patient was diagnosed with suspected brown tumor secondary to SHPT. Maxillofacial involvement in brown tumors is rare and may mimic other giant cell lesions radiologically and histologically, making diagnosis challenging; however, markedly elevated iPTH levels strongly support the diagnosis. This case highlights the importance of a multimodal diagnostic approach and early monitoring of mineral and bone metabolism in long-term hemodialysis patients to improve outcomes.

Keyword: Brown tumor; Maintenance hemodialysis; Secondary hyperparathyroidism; Panoramic radiography

INTRODUCTION

Brown tumor is a rare, benign, and erosive focal bone lesion characterized by increased osteoclastic activity and fibroblastic proliferation resulting from uncontrolled hyperparathyroidism[1,2]. As a severe manifestation of renal osteodystrophy within the spectrum of chronic kidney disease-Mineral and Bone Disorder (CKD-MBD), these lesions are driven by the overproduction of parathyroid hormone (PTH)[3,4,5]. In patients with end-stage renal disease, particularly those undergoing maintenance hemodialysis, chronic imbalances in calcium, phosphorus, and vitamin D metabolism trigger persistent PTH elevation. Nearly 90% of CKD patients on hemodialysis develop secondary hyperparathyroidism due to excessive PTH secretion[6]. This hormonal excess promotes progressive bone resorption and high-turnover bone disease, ultimately leading to the formation of brown tumors.

The incidence of brown tumors as a complication of hyperparathyroidism is remarkably low at approximately 0.1%, though it increases to 1.5–1.7% in patients with CKD-associated secondary hyperparathyroidism[1,7,8]. While these lesions typically involve the axial skeleton and long bones, they can manifest in any osseous region. Maxillofacial involvement is particularly uncommon, accounting for only 4.5% of cases, with a predilection for the mandible over the maxilla. Furthermore, this condition demonstrates a significant demographic bias toward women over the age of 50, with a reported female-to-male ratio of 3:1 [3,8-12].

Establishing a diagnosis of brown tumor in hemodialysis patients remains clinically challenging, as radiographic and histological features frequently mimic other giant-cell bone lesions. Consequently, neither imaging nor histopathology can serve as a standalone diagnostic modality; rather, a definitive diagnosis necessitates a multidisciplinary approach that integrates clinical, biochemical, and radiological findings[3,12]. Herein, we report a rare and diagnostically complex case of a suspected brown tumor in a young woman undergoing maintenance hemodialysis, highlighting a presentation that deviates from established demographic norms.

CASE ILLUSTRATION

A 28-year-old woman presented to the Emergency Department of Dr. Soedono General Hospital with a one-week history of dyspnea, which exacerbated post-hemodialysis, accompanied by a productive cough and fever. The patient also reported a progressively enlarging, painless mass on the palate that had persisted for one year, significantly obstructing mastication and speech. The mass was non-ulcerated, showed no signs of hemorrhage,

and was not associated with dental or other intraoral symptoms. Notably, the patient experienced a marked 13 cm reduction in height (from 158 cm to 145 cm) over the previous year. Her medical history was significant for end-stage renal disease, for which she had been undergoing maintenance hemodialysis twice weekly for seven years. Additional comorbidities included hypertension, cardiomegaly, and a previous pericardial effusion requiring drainage one year prior. Clinical history revealed anuria, while current medications included amlodipine, bisoprolol, methyldopa, folic acid, and sodium bicarbonate; however, the patient reported poor adherence to calcium carbonate supplementation.

Physical examination revealed a patient in a weakened state but with a clear sensorium (compos mentis). Vital signs were as follows: blood pressure 143/70 mmHg, pulse 99 beats/minute (regular and strong), respiratory rate 26 breaths/minute, and an axillary temperature of 37.4°C. Oxygen saturation was 95% while receiving supplemental oxygen via a non-rebreathing mask (NRM). Head and neck assessment showed conjunctival pallor and elevated jugular venous pressure. Thoracic examination revealed symmetrical chest expansion without retractions; however, cardiac percussion indicated cardiomegaly, with the right heart border widened and the ictus cordis displaced to the fifth intercostal space (ICS V) at the left anterior axillary line. Abdominal and extremity examinations were unremarkable. Intraoral evaluation identified a firm, solid mass on the hard palate, approximately 4 cm in diameter, with no evidence of erosion, suppuration, or hemorrhage. A chest X-ray confirmed cardiomegaly and bilateral pulmonary infiltrates. The results of the physical examination and chest X-ray can be seen in Figure 1.

Figure 1. The results of the physical examination and chest X-ray

Initial laboratory findings were significant for anemia and renal dysfunction: hemoglobin 8 g/dL, leukocyte count 11,220/mm³, neutrophils 70.1%, and platelet count 155,000/mm³. Biochemical analysis showed blood urea nitrogen (BUN) 30.4 mg/dL, creatinine 4.3 mg/dL, random blood glucose 112 mg/dL, and albumin 3.62 g/dL. Electrolytes were within normal limits (sodium 137 mmol/L, potassium 3.5 mmol/L, chloride 103 mmol/L). Arterial blood gas (ABG) analysis under NRM oxygen indicated respiratory acidosis: pH 7.27, PCO_2 69 mmHg, PO_2 82 mmHg, and HCO_3^- 31.4 mmol/L.

The patient is diagnosed with CKD on a maintenance hemodialysis schedule every Tuesday and Friday, currently presenting with respiratory failure, partially compensated respiratory acidosis, pneumonia, and suspected pulmonary tuberculosis. Physical examination also revealed a palatal mass suspicious for a brown tumor; a localized bone lesion associated with secondary hyperparathyroidism. To further evaluate these conditions, the patient is scheduled for a diagnostic workup including a GeneXpert TB test, sputum culture, and laboratory assessments of calcium, iPTH, alkaline phosphatase, and Vitamin D levels. Imaging studies, specifically panoramic radiography and a non-contrast maxillofacial CT scan, will be performed once the patient reaches clinical stability, followed by a Fine Needle Aspiration Biopsy (FNAB). Despite clinical severity, the patient's family has refused ICU or HCU admission; consequently, the patient is being managed in a dedicated tuberculosis ward. Initial therapy consists of a moxifloxacin drip (400 mg once a day), amlodipine (10 mg once a day), methyldopa (500 mg twice a day), folic acid (400 mcg twice a day), and calcium carbonate (500 mg twice a day), alongside the continuation of the prescribed hemodialysis sessions.

Panoramic radiography revealed an expansive lytic (radiolucent) lesion in the palate, extending predominantly toward the right maxilla. A head CT scan has not yet been performed due to the patient's clinical instability. On the third day of hospitalization, laboratory results showed calcium at 8.4 mg/dL, vitamin D at 14.4 ng/mL, phosphorus at 5.4 mg/dL, alkaline phosphatase at 1,721 U/L, and iPTH at 3,239 pg/mL. Additionally, the GeneXpert test detected Mycobacterium tuberculosis (rifampicin-sensitive). These radiographic and laboratory findings further support the diagnosis of a brown tumor secondary to hyperparathyroidism. Consequently, the patient was also diagnosed with secondary hyperparathyroidism and pulmonary tuberculosis. Treatment was initiated with vitamin D at 5,000 units once daily, and the patient is scheduled to begin anti-tuberculosis therapy.

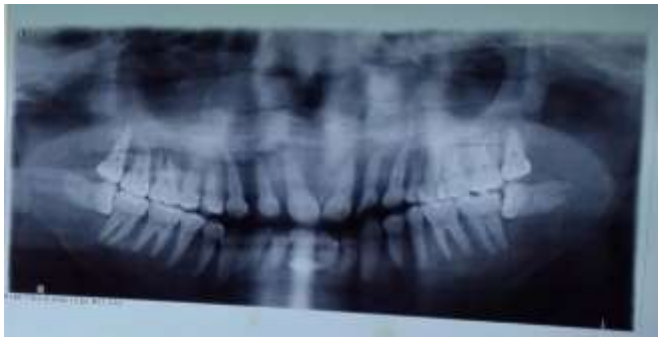

Figure 2. The results of Panoramic radiography

On the fourth day of hospitalization, FNAB results revealed a distribution of erythrocytes, suggestive of a vascular lesion. As these results did not show the presence of giant cells, a confirmatory tissue biopsy was planned. However, shortly before the scheduled hemodialysis session later that day, the patient's condition acutely deteriorated, and they were subsequently pronounced dead due to respiratory failure. Based on the comprehensive clinical workup, the final diagnosis was established as CKD on maintenance hemodialysis, suspected brown tumor, secondary hyperparathyroidism, pulmonary tuberculosis, and respiratory failure.

DISCUSSION

In patients undergoing long-term hemodialysis, disturbances in mineral metabolism such as hyperphosphatemia, hypocalcemia or normocalcemia, and vitamin D deficiency commonly occur. These abnormalities stimulate the parathyroid glands to increase PTH secretion to maintain calcium homeostasis, primarily by mobilizing calcium from the bones into the bloodstream. In addition, hyperphosphatemia promotes the elevation of fibroblast growth factor 23 (FGF23), which further suppresses the production of 1,25-dihydroxyvitamin D. This pathophysiological process is referred to as secondary hyperparathyroidism, a common complication observed in patients with advanced CKD[10,13,14].

Secondary hyperparathyroidism and persistently elevated FGF23 levels may eventually result in CKD-MBD, a condition characterized by abnormalities in mineral and bone metabolism. This disorder promotes osteoclastic activity over osteoblastic activity, accompanied by fibroblastic proliferation, leading to renal osteodystrophy. Increased bone resorption causes destruction of bone tissue, particularly along the periosteal and endosteal surfaces, which are subsequently replaced by fibrous tissue and associated with reduced bone mineral density. Consequently, skeletal abnormalities such as brown tumors may develop, rendering the bones more fragile and increasing the risk of height loss and fractures[3,14-17]. Furthermore, activation of the renin–angiotensin–aldosterone system and proinflammatory cytokines, including IL-1α, IL-6, and TNF-α, also contribute to enhanced osteoclastic activity in CKD[3]. Brown tumors are generally painless lesions presenting as masses that may arise

in any bone; however, they are uncommon in the maxillofacial region and are more frequently observed in women and older individuals (>50 years)[11,18].

Brown tumor can be diagnosed using panoramic radiography, CT scan, or MRI. Panoramic imaging typically demonstrates well-defined osteolytic (radiolucent) lesions, while CT scan may reveal lytic lesions with a ground-glass appearance. MRI can further identify mass-like changes in the affected bone[1,7,19]. Similar radiologic findings may also be seen in giant cell tumors, central giant cell granulomas, aneurysmal bone cysts, bone metastases, and osteosarcomas. However, osteosarcomas more commonly involve long bones, whereas brown tumors are associated with markedly elevated PTH levels[3,8,19,20]. Table 1 summarizes the radiologic differences between brown tumors and other bone lesions.

Table 1. Difference between brown tumors and other bone lesions based on radiology

	BROWN TUMOR	FIBROUS DYSPLASIA	GIANT CELL TUMOR	ANEURYSMAL BONE CYST	LYTIC METASTASES	MULTIPLE MYELOMA

X-ray	Well defined lytic lesions, radiolucent; thin or broken cortex; X-ray signs of PHPT	Focal lytic, mixed or sclerotic lesion	Solid or mixed well defined solid-cystic lesion; bone expansion; cortical thinning; non-sclerotic margins	Focal, multi lobulated, pseudocyst; lytic lesions; thin sclerotic margins	Focal, lytic lesions; bone destruction; narrow zones of transition; periosteal reaction	Lytic expansive lesions; prominent cortical thinning; well defined, non-sclerotic margins
CT	Irreguler, multi-loculated lytic lesions; 'ground glass opacification " and interruption of the cortex; contrast enhancement	Ground-glass matrix; mixed pattern of ground glass, lytic or sclerotic appearance	Solid or mixed solid-cystic lesion; bone expansion; hyper-dense solid components with contrast enhancement	Focal, multi-lobulated, pseudocyst; lytic lesions with fluid-fluid levels; cortical thinning; no contrast enhancement	Lytic lesions; poorly defined margins; bone destruction	Punched-out expansive lytic lesions
MRI	Isohypo-intense signal in T1,T2-weighted sequences; contrast enhancement of the solid portions and septa of cystic lesions; T2 hyperintense in cystic lesions and contrast enhancement from the periphery and septa	Low signal intensity on T1-T2 weighted images in ossified or fibrous portions; heterogenous enhancement and signal intensity in the active phase	Hypo-isointense signal on T1-weighted sequence; hypo-intensity on T2; heterogeneous contrast enhancement	Different T1 and T2 intensity signal of intra-cavitary fluid-fluid levels; surrounding rim of low T1 and T2 signal possible septa enhancement	Soft hypo-intense tissue on T1 replacing bone marrow; heterogeneous contrast enhamcement	Hypo-isotense signal on T1; hyper-intense signal on T2 fat suppression sequences; early contrast enhancement
Bone scintigraphy	Increased focal uptake	Increased focal uptake	Increased periferic uptake with central photopenia ("donut sign")	Homogenous increased / mild uptake	Increased focal or diffuse uptake	Increased uptake
Total calium	High	Normal	Normal	Normal	High	High/Normal
PTH	High	High/Normal	Normal	Normal	Low	Low/Normal
Alkaline phosphatase	High	High/Normal	Normal	Normal	High	Normal

The patient presented with a palatal mass, height loss, and laboratory findings of hypocalcemia, hyperphosphatemia, vitamin D deficiency, and elevated iPTH levels. Panoramic radiography demonstrated a radiolucent lytic lesion involving the palate and extending predominantly to the right maxilla. These findings strongly suggest secondary hyperparathyroidism with a suspected brown tumor, warranting further histopathological evaluation.

Histopathological examination is required to confirm brown tumor diagnosis. FNAB provides a faster and less invasive alternative to excisional or incisional biopsy, although the latter remains more definitive. Brown tumors typically show multinucleated giant cells, spindle stromal cells, hemorrhagic areas, and hemosiderin deposits, reflecting their highly vascular nature[8,13,21]. Because similar histologic features may also occur in giant cell tumor of bone, central giant cell granuloma, and aneurysmal bone cyst, histopathology alone is insufficient for diagnosis. Brown tumors are distinguished primarily by laboratory findings, particularly elevated iPTH with abnormalities in calcium and phosphate levels. Therefore, accurate diagnosis requires integration of clinical, laboratory, radiologic, and histopathologic findings[3,20,21]. The radiologic, laboratory, and histopathologic differences between brown tumors and other bone lesions are summarized in Table 2.

Table 2. Difference between brown tumor and other bone lession

	BROWN TUMOR	CENTRAL GIANT CELL GRANULOMA	GIANT CELL BONE TUMOR
Medical history	Primary HPT: nephrolithiasis, muscle weakness, osteoporosis, abdominal complaints, and psychiatric symptoms. SecondaryHPT: renal osteodystrophy, which can result in bone loss, bone pain,and fractures	Non-specific pain and swelling that may precede diagnosis by months to years	10% to 20% present with a pathological fracture. Rarely multifocal. Malignant transformation in only up to 1% of cases. Only up to 4% of patients develop pulmonary metastases
Patient features	Female: male ratio 2.8:1 Mean age 42 years (11-83)	2nd and 3rd decades of life. Female:male ratio 2:1	Skeletally mature patients, usually 30 to 50 years old, slight female predominance
Clinical picture	Pathologic fractures, local bone pain. 65% of tumours occurred in the face; of these, 48% in the mandible, 38% in the maxilla, and 14% involved both jaws	Localised tumours (2) most often found in the maxilla, otherwise in the mandible. Frequently only a painless swelling, in some cases rapid growth and bone erosion by the mass, particularly on the alveolar ridge	Several months of pain, local swelling, and limited range of motion of the adjacent joint. Usually found in long bones, only 10% occur in the axial skeleton
Biochemical profile	Elevated PHT Primary HPT: hypercalcaemia, hypophosphatemia (1). Secondary HPT: normo- or hypocalcaemia, normo- or hyperphosphatemia	Normal PTH, normocalcaemia, normophosphatemia.	Normal PTH, normocalcaemia, normophosphatemia

Radiographic aspect	Well defined, uni- or multilocular lytic expanding defect which may expand or erode periosteum. Concomitantly appearing subperiosteal resorption	Lysis with expansile remodeling of bone and a multilocular appearance. Thin, usually intact cortex	Eccentric, lytic lesion with a nonsclerotic and sharply defined geographic border. Centered in the metaphysis with extension to subchondral bone. Soft tissue extension is commonly present
Macroscopic morphology	Haemorrhage and hemosiderin deposition cause reddish brown colour of the tissue specimen (2). Often undergoes central degeneration and exhibit fibrous marrow replacement	Lobulated mass of proloferative vascular connective tissue	Large, well vascularised lesions with broad bands of collagenous fibrous tissue, eccentrically located in metaepiphyseal bone and extending to the articular surface. Cortical bone usually expanded
Histologic morphology	Dense, spindle cell stroma, focal areas of osteoid, cystic degeneration, microfractures, hemorrhage, hemosiderin-laden macrophages, and multinucleated giant cells or osteoclasts	Granulomatous appearance produced by plump, bland fibroblasts. Multinucleated giant cells (osteoclasts) often focal in distribution around multiple foci of haemorrhage, along which osteoid is produced. Fibrous matrix, occasional trabeculae of woven bone. Extension to subchondral bone only rare	Giant cell tumours are believed to result from an over-expression in the RANK/RANKL signalling pathway with resultant over-proliferation of osteoclasts. Rounded mononuclear macrophage-like osteoclast precursor cells (neoplastic), spindle-shaped mononuclear “stromal” cells (neoplastic), Lacunar bone resorption, absent matrix mineralisation
Recurrence	Dependant of parathyroid disease state	Reported but rare	27%–65% after isolated curettage, 12%–27% after curettage with adjuvants, 0%–12% after en bloc resection (10). Denosumab an monoclonal antibody anti-RANKL is a potential adjuvant therapy.

In this patient, FNAB revealed a distribution of erythrocytes, suggesting vascular lesion. No giant cells were identified, which may have been due to suboptimal technique during the FNAB sampling process. Nevertheless,

based on the impression of a vascular lesion, the palatal mass was highly suspected to represent a brown tumor. A confirmatory tissue biopsy had been planned; however, it was not performed because the patient died. Based on the combined clinical, laboratory, and radiological findings, the patient was diagnosed with suspected brown tumor of the palatal region due to secondary hyperparathyroidism.

CONCLUSION

We describe a rare and diagnostically complex case of a 28-year-old woman receiving maintenance hemodialysis who presented with a suspected brown tumor in the palatal region secondary to hyperparathyroidism. The diagnosis was established through the integration of clinical presentation, laboratory abnormalities, and radiologic findings. Because the radiologic and histologic characteristics of brown tumors can mimic other bone lesions, definitive diagnosis requires careful clinical correlation, particularly with laboratory parameters. Prompt recognition of this condition is important, as early treatment of hyperparathyroidism, either medically or surgically, may lead to better clinical outcomes.

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