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Case Report

Brown tumor of the rib associated with primary hyperparathyroidism misdiagnosed as metastatic deposit - A case report

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ABSTRACT

Brown tumor is a rare skeletal manifestation where multiple giant cell rich bony lesions, arise as a direct result of either primary or secondary hyperparathyroidism. It is caused by excess osteoclastic activity and hemosiderin deposition because of uncontrolled parathormone secretion. Brown tumors usually involve mandible, maxilla, clavicle, ribs, and pelvic bones. Our case report describes the case of a 42-year-old lady who consulted our hospital for multiple bony lesions in ribs suspicious of metastasis and summarizes our experience in diagnosis of similar cases that we might come across in the future.

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1. Introduction

Brown tumor, is benign yet unusual giant cell tumor of hyperparathyroidism. It represents a reparative process, rather than a neoplastic origin, as a result of disturbed bone remodeling, due to long-standing increase in parathormone level. Large amount of calcium from bones is mobilized into the blood flow, resulting in fibrous tissue hyperplasia in the bones with leaky hemorrhage. Then reactive macrophage hyperplasia occurs which phagocytose the red blood cells ultimately forming a brown nodule.

2. Case Presentation

A 50-year-old woman from central India presented with back pain since last one year. Radiological examination showed ill-defined expansile lytic area in right 7th rib and bilateral lamina of D4 vertebral body on CT scan. Further biopsy revealed giant cell containing tumour consisting of variable proportion of small, round to oval and spindled mononuclear cells having nuclei with dispersed chromatin,

small nucleoli and pale eosinophilic cytoplasm admixed with plenty multinucleated osteoclastic giant cells, few bony trabeculae with resorptive changes and numerous pigment laden macrophages. Giant cell rich lesion along with multiple lytic lesion of rib and D4 vertebra favored brown tumor of hyperparathyroidism and the patient was advised for further biochemical investigation that revealed serum alkaline phosphatase 492 IU/L, serum PTH levels 881.7 pg/ml, serum calcium level 14.0 mg/dl and serum inorganic phosphorous 2.4 mg / dl. Final diagnosis of brown tumor of hyperparathyroidism was made and patient was advised endocrinology consultation.

3. Discussion

Brown tumor is not a true neoplasm. It is a rare presentation of either primary or secondary hyperparathyroidism. Also called von Recklinghausen disease of bone. The name “brown tumor” derives from the color, which is caused by the vascularity, hemorrhage and deposits of hemosiderin. Brown tumors do not usually present with pain unless it compresses on neighbouring neural structures, so most often they are discovered incidentally. Elevated parathormone

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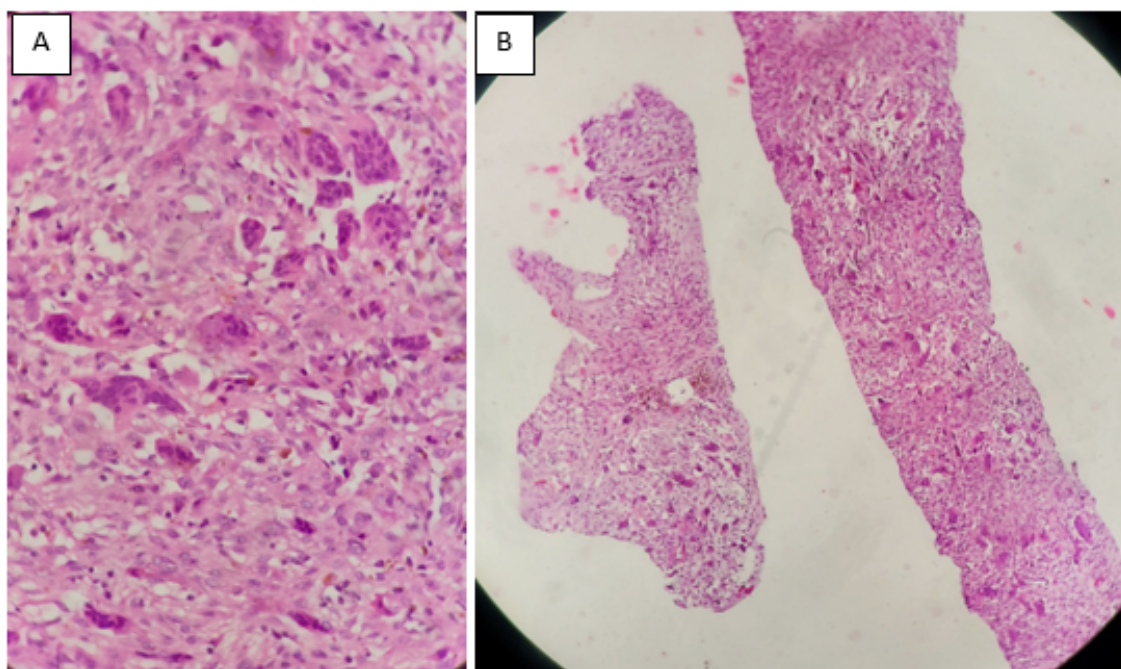


Fig. 1: Tumour containing numerous multinucleated osteoclastic type giant cells admixed with small round to oval, often spindled mononuclear cells with dispersed chromatin, small nucleoli and eosinophilic cytoplasm with adjacent bony trabeculae with resorptive changes. (A: H&E 10X; B: H&E 4X)

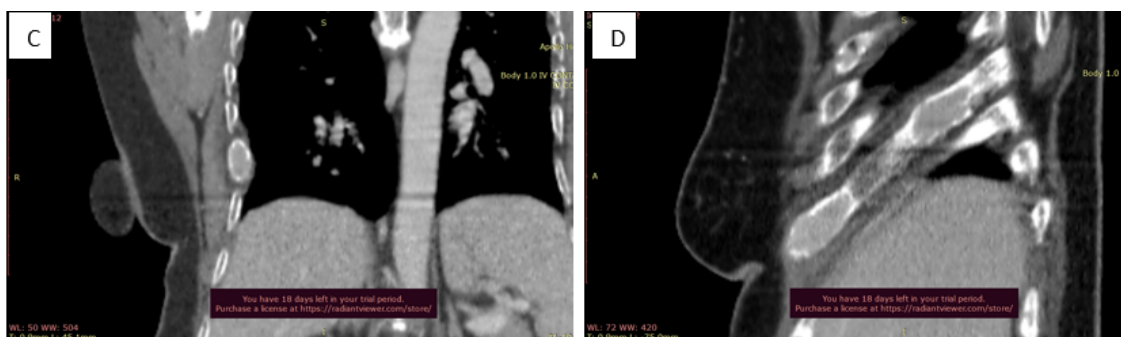


Fig. 2: Ill defined expansile lytic area in right 7th rib and bilateral lamina of D4 vertebral body.

level results in the abnormal bone remodeling which brings about unusual reactive lesion. It is an extreme form of osteitis fibrosa cystica.¹ The reported prevalence of brown tumor is 0.1%.² They have been reported to occur in 4.5% of patients with primary hyperparathyroidism and 1.5 to 1.7% of those with secondary disease.^{3–5} Histologically brown tumors are identical to giant cell tumor and therefore, this entity can easily be misdiagnosed if close attention is not paid to the clinical presentation, biochemistry and radiographic features. Giant cell tumor (osteoclastoma) and brown tumor of parathyroid are very much similar, so suspicion should arise while making the final diagnosis. The diagnosis requires a systemic investigation and array of laboratory studies that include serum calcium, phosphorus and alkaline phosphate. Hyperparathyroidism can be

primary, secondary or tertiary. Primary hyperparathyroidism is characterized by uncontrolled parathormone production and hypercalcemia, mostly due to adenomatous lesion of parathyroid. Secondary hyperparathyroidism is usually caused by vitamin D deficiency, malabsorption, or due to hypercalciuria. Low serum calcium levels resulting from primary diseases brings about redundant secretion of parathormone. Tertiary hyperparathyroidism usually develops from secondary hyperparathyroidism and evolves into a more severe circumstance with autonomous parathormone secretion.⁶ The management of brown tumor mainly focuses on correction of the underlying disorder and maintenance of normal parathormone and serum calcium levels. Use of systemic or intralesional corticosteroid have been reported to reduce the size of the lesion. Surgical

excision is only indicated in non responders and in large lesion causing severe disfigurement.⁷ Long-term follow-up of such lesion is mandatory as variable clinical behavior of the lesion following normalization of the PTH and serum calcium levels has been reported.⁸ Giant cell tumor of the bone, solid aneurysmal bone cyst, and giant cell reparative granuloma are included in the differential diagnoses for Brown tumors. They might cause pain and microfractures in any part of the skeletal system. Hyperparathyroidism may cause renal failure and kidney stones.

4. Conclusion

Brown tumour, although rare is often difficult to differentiate from osteoclastoma or giant cell tumour of bone. The symptoms frequently overlap and it is essential to keep in mind the exclusive sites of these lesions to successfully make the final diagnosis. Histologically, it is impossible to distinguish a Brown tumor of hyperparathyroidism from other giant cell lesions of bone. Enhanced CT scan, laboratory examination, ultrasonography, ^{99m}Tc-MIBI SPECT-CT scintiscan and pathological examination along with biochemical tests are needed to confirm the diagnosis of brown tumor.

5. Conflict of Interest

None.


6. Source of Funding


None.


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