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RESEARCH ARTICLE
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A FAUX TUMOUR OF THE CANCER

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Abstract
Browns tumour is a rare tumour like lesion of the bone, which is considered as an end stage lesion of abnormal bone metabolism caused by persistently high parathyroid hormone levels. These are highly vascular osteopathic lesions. In hyperparathyroidism patients instead of neoplasticism it depicts a reparable cellular process. Either these tumours can be aggressive or destructive. It can be seen in any part of the skeleton. Sometimes it can be seen multiple bones and misdiagnosed as metastatic tumour.
Key words: Parathyroidism, Browns tumour, Parathyroid hormone

Introduction
Primary hyperparathyroidism(1) is a disorder in which high parathyroid hormone is released from parathyroid glands. This increased PTH levels results in defective calcium levels, phosphate levels and metabolism of bone which results in hypercalcemia(8) and hypophosphatemia. A brown tumour(5) is a benign , focal bony lesion due to rapid osteoclastic activity of bone. It is one of the most pathognomic signs of primary hyperparathyroidism.
It is seen in 1.5 percent of patients with hyperparathyroidism(3) and 4.5 percent of patients with secondary hyperparathyroidism. Primary hyperparathyroidism is more commonly seen in woman than man. Usually Incidence occurs in 6th/7th decade of life(2).

Case report
We report a case of 45 year old female with c/o multiple joint pains since 6 months which is insidious in onset, gradually progressive. She also had complains of generalised tiredness since 6months. Patient is a known case of hypothyroidism and on regular treatment. On general examination patient was conscious, oriented, afebrile, pulse rate- 66/min blood pressure-110/80 mmhg. On examination pallor present, bilateral pitting pedal oedema present, no icterus, cyanosis. Cardiovascular, respiratory, abdominal, neurological examinations were normal. Local examination shows small joint tenderness, elbow joint, wrist joint, knee and ankle joint tenderness.

All necessary investigations were done. Suspected Rheumatoid arthritis, immunological profile was sent in which anti CCP-16.5 negative, ANA profile negative, CRP-
0.4(negative) RA factor came to be negative. X-ray hand and X-ray spine was taken which shows-osteolytic lesions. Parathyroid hormone levels and serum calcium was sent showed serum calcium-15.8, parathyroid hormone-2292, serum phosphorous- 2.0. Based on these values diagnosis was made as primary hyperparathyroidism. Further investigations are as follows , CT brain was done which shows bony expansion with cortical thinning and diffuse lyric areas giving salt and pepper appearance, and evidence of well defined expansive lytic lesion in the proximal ramus of mandible on left side likely brown tumour measuring nearly1.1*1.6*2cms. MRI abdomen was done which showed evidence of well defined expansive lytic lesion noted at D8 vertebral body. Endocrinologist opinion obtained in view of hyperparathyroidism, suggested to do sestamibi scan which showed right superior parathyroid adenoma. Following management of bone pain, Calcium correction was done by giving IV fluids and Inj.Calcitonin subcutaneous. Surgery opinion obtained in view of parathyroid adenoma after calcium correction. Right superior parathyroidectomy was done . Post operative histopathological examination revealed features of a parathyroid adenoma. Four days postoperatively serum PTH level was decreased to 30.2pg/ml. Now patient is stable symptoms improved symptomatically and clinically. Serum calcium and parathyroid hormone levels came to be normal and patient was discharged.

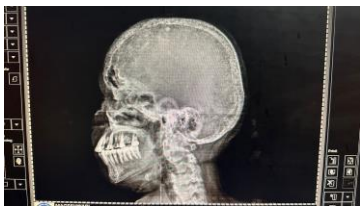

Fig 1. X-ray Skull showing SALT AND PEPPER appearance

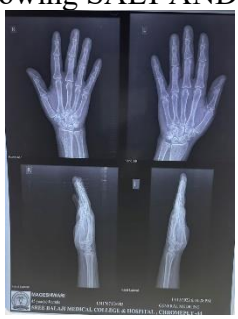

Fig 2. X-ray hand showing osteolytic lesions

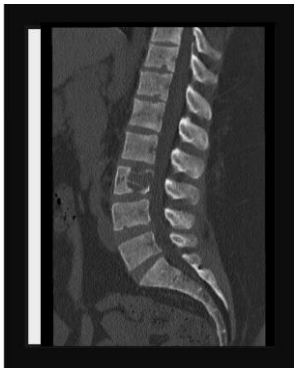

Fig 3. Xray LS spine showing BROWNS tumour

Discussion

Primary hyperparathyroidism is a disorder in which high parathyroid hormone is released from parathyroid glands. The most common clinical presentation of primary hyperparathyroidism is asymptomatic hypercalcemia. Atypical presentation include normocalcemic PHPT and parathyroid

crisis. The classical manifestations of PHPT include bones, stones, abdominal moans and psychic groans. They reflect combined effects of increased PTH secretion and hypercalcemia. The abnormalities associated with hyperparathyroidism are nephrolithiasis and bone disease, both are due to prolonged PTH excess. Symptoms attributable to hypercalcemia include anorexia, nausea, constipation, polydipsia and polyuria. The most common symptoms at presentation include bone pain(56percent), renal calculi(31present), and weakness/
fatiguability(59percent). The classic presentation of PHPT bone disease is osteitis fibrosa cystica, which is clinically characterised by bone pain and subperiosteal bone resorption on radial aspect of middle phalanges, tapering of distal clavicles, a salt and pepper appearance of skull and brown tumour of long bones. Browns tumour result from excess osteoclastic activity and consists of collections of osteoclasts intermixed with fibrous tissue and poorly mineralised woven bone. The brown colour is due to hemosiderin deposition. It is uncommon and pathognomic sign of hyperparathyroidism. These tumours usually present as solitary lesion, localised to the facial bones and also most commonly seen in secondary hyperparathyroidism than primary hyperparathyroidism.
Our case is unique, as the patient is presenting with multiple brown tumours. The presentation of our case is atypically severe, we could have considered another possible diagnosis. The main differential diagnosis are malignancy, familial hypocalciuric hypercalcemia- an autosomal dominant disorder, drugs usage like thiazide and lithium, secondary hyperparathyroidism. Severe classical PHPT is associated with increased mortality, primarily due to cardiovascular disease. Some studies described that mortality rate can be declined with time from parathyroidectomy.

References

1. Sun B, Guo B, Wu B, et al. Characteristics, management, and outcome of primary hyperparathyroidism at a single clinical center from 2005 to 2016. Osteoporos Int 2018; 29:635.
2. Lazaretti-Castro M. The diagnosis of primary hyperparathyroidism in developing countries remains in the past century: still with bones, stone and groans. Arch Endocrinol Metab 2020; 64:101.
3. Yadav SK, Johri G, Bichoo RA, et al. Primary hyperparathyroidism in developing world: a systematic review on the changing clinical profile of the disease. Arch Endocrinol Metab 2020; 64:105.
4. Arya AK, Kumari P, Bhadada SK, et al. Progressive rise in the prevalence of asymptomatic primary hyperparathyroidism in India: Data from PHPT registry. J Bone Miner Metab 2021; 39:253.
5. Mithal A, Kaur P, Singh VP, et al. ASYMPTOMATIC PRIMARY HYPERPARATHYROIDISM EXISTS IN NORTH INDIA: RETROSPECTIVE DATA FROM 2 TERTIARY CARE CENTERS. Endor Pract 2015; 21:581.
6. Silverberg SJ, Bilezikian JP. Evaluation and management of primary hyperparathyroidism.] Clin Endocrinol Metab 1996; 81:2036.
7. Silverberg SJ, Clarke BL, Peacock M, et al. Current issues in the presentation of asymptomatic primary hyperparathyroidism: proceedings of the Fourth International Workshop. J Clin Endocrinol Metab 2014; 99:3580.
8. Siperstein AE, Shen W, Chan AK, et al. Normocalcemic hyperparathyroidism. Biochemical and symptom profiles before and after surgery. Arch Surg 1992; 127:1157.