Case Report

Parathyroid Adenoma: Sand Behind the Storm

Neha Bagri[1], Ritu Misra[2], Geetika Sindhwani[3], Anuradha Sharma[4]

[1,3,4]Assistant Professor, [2]Professor and Head, Department of Radiodiagnosis, VMMC and Safdarjung Hospital, New Delhi, India

DOI:10.37821/ruhsjhs.5.3.2020.240

ABSTRACT

Hyperparathyroidism is a metabolic disorder caused by increased levels of parathormone levels. Depending on underlying etiology, it can be sub-classified into primary, secondary, and tertiary types. Hyperparathyroidism can present as hyperplasia or adenoma in association with syndromes like Multiple Endocrine Neoplasia (MEN) or other familial syndromes. We are presenting a case of hyperparathyroidism due to underlying parathyroid adenoma with characteristic radiological and nuclear imaging findings.

Keywords: Nuclear imaging, Parathyroid adenoma.

INTRODUCTION

Hyperparathyroidism can be classified into primary, secondary, or tertiary hyperparathyroidism depending on the cause. Primary hyperparathyroidism is due to hypersecretion of parathormone (PTH) from primary parathyroid gland lesions, usually occurs due to parathyroid adenoma (80%) but can also be caused by parathyroid gland hyperplasia (15%-20%) or carcinoma (0.5%).[1] Secondary hyperparathyroidism results from decreased calcium levels (as in chronic renal failure) or insensitivity of parathyroid glands (pseudo-hypoparathyroidism) to elevated serum calcium levels causing stimulation of the parathyroid glands. Tertiary hyperparathyroidism is seen in cases of secondary hyperparathyroidism in which the parathyroid glands continue to function autonomously despite correction of the initial cause, resulting in hypersecretion of PTH in the setting of normal calcium levels.[1,2] We are reporting a case of young female with parathyroid adenoma presenting with bony lesions and diffuse marrow changes.

Case Description: A 28 years old female patient presented with pain and swelling in left knee since 4 months. Plain radiograph (antero-posterior view) of bilateral knees anteroposterior view revealed a well-defined expansile lytic lesion with narrow zone of transition in the distal epimetaphyseal region of left femur with associated pathological fracture and mild medial subluxation of proximal femur. Lesion was not extending upto the articular surface. There was another small lytic lesion with marginal sclerosis in the medial cortex of mid-shaft of left femur (Figure 1).

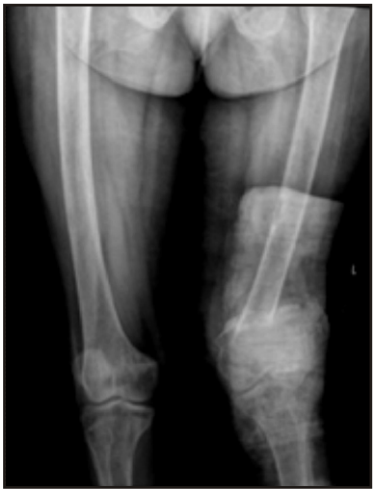
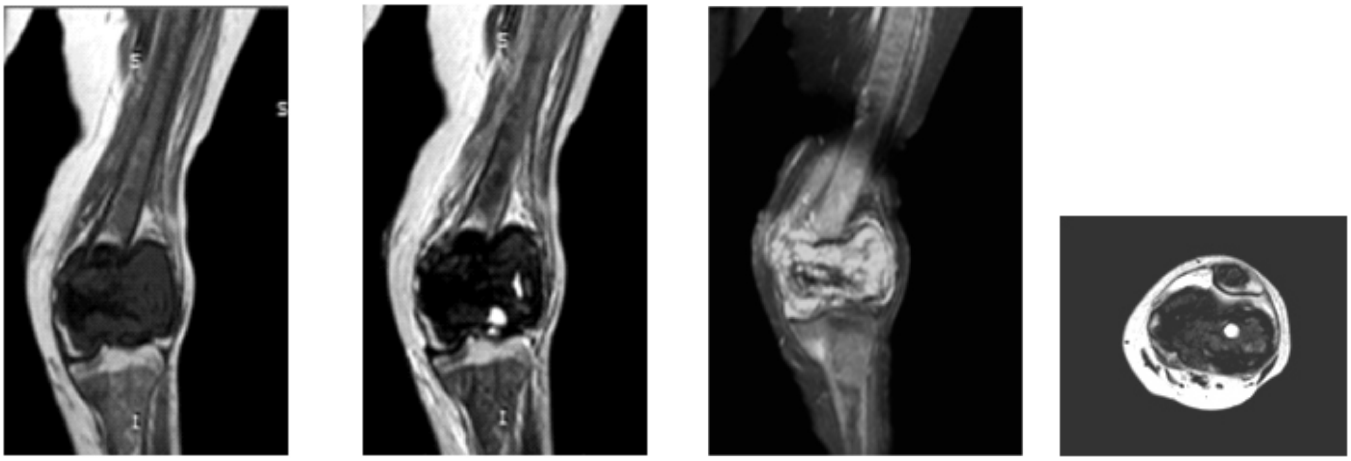


Figure 1: Plain radiograph bilateral thighs with knee (AP view).

On MRI knee, a well defined lesion was seen in epimetaphyseal region of left femur, which was hypointense signal on T1W and T2W sequences, and showed mixed signal intensity on STIR. Few T2 hyperintense cystic areas were also seen in the lesion and gradient sequence revealed areas of blooming in the lesion. The lesion showed heterogeneous contrast enhancement. No evidence of extension to joint or surrounding soft tissues was seen. There were other similar appearing lesions in femoral diaphysis and patella. There was marrow signal alteration (T1 and T2 hypointense) in visualized bones (Figure 2). In view of marrow alteration and suspicion of some metabolic

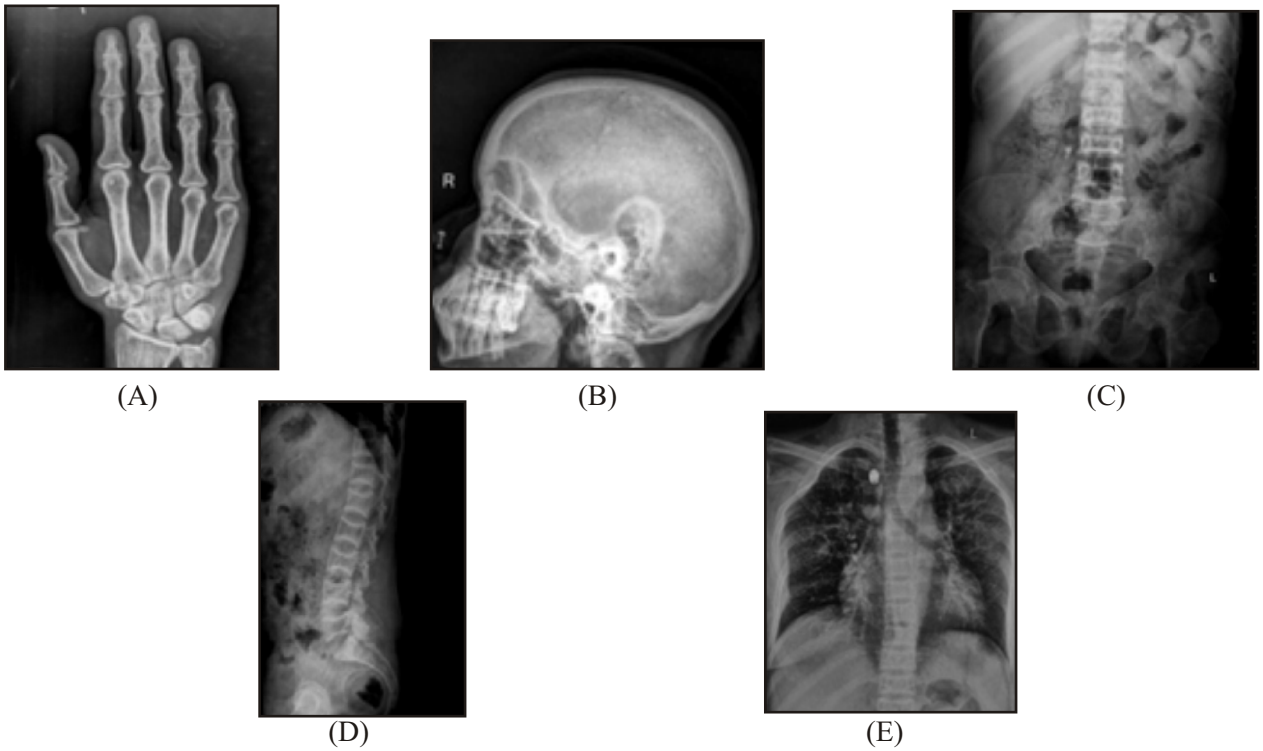


(A) (B) (C) (D)

Figure 2: T1 W coronal (A), T2 W coronal (B), Contrast coronal (C), and T2W axial (D) MRI knee reveals T1 and T2 hypointense lesion in left femoral epi-metaphyseal location reaching upto articular surface with few cystic areas. Diffuse marrow alteration is seen in visualised bones. Other similar appearing lesions are seen in patella and femoral diaphysis.

bone disease, skeletal survey of patient was performed (Figure 3).

There was diffuse osteopenia of visualized bones, subperiosteal erosions in radial aspects of phalanges, and acro-osteolysis in distal phalanges and was classical salt and pepper appearance in skull. Biconcave vertebrae and features of triradiate pelvis were noted, suggestive of severe osteopenia. Radio-opacity at right L4-L5 intervertebral disc level was also seen. There was presence of multiple pathological fractures in ribs with multiple osteolytic lesions. On Tc-99m MDP bone scan, there was superscan with non-uptake of contrast in soft tissues and



(A) (B) (C)

(D) (E)

Figure 3: Diffuse osteopenia in visualised bones. Subperiosteal erosions and acroosteolysis in hands (A), Salt-Pepper skull (B), Right upper ureteric calculus (C), Biconcave vertebrae (D), Multiple brown tumours with rib fractures (E).

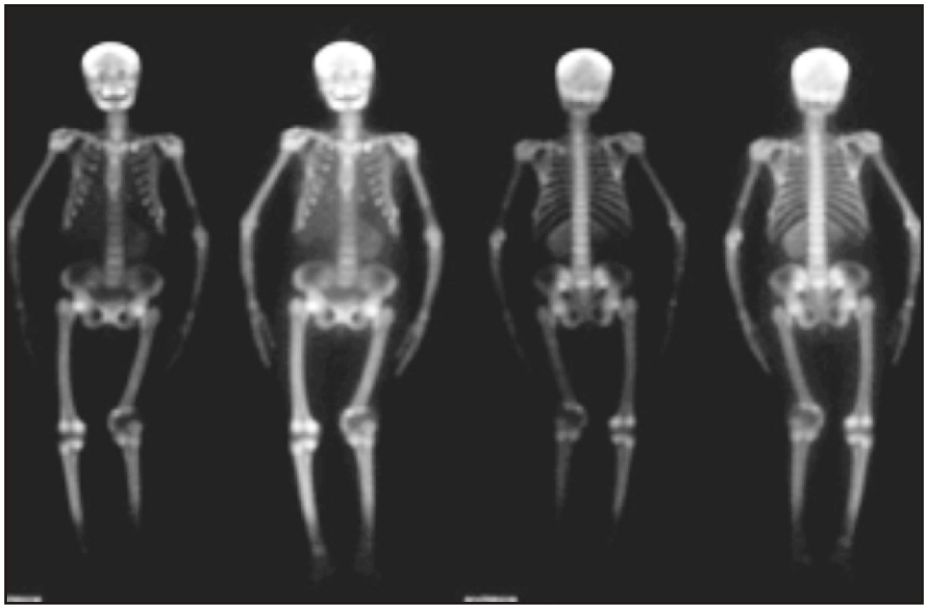
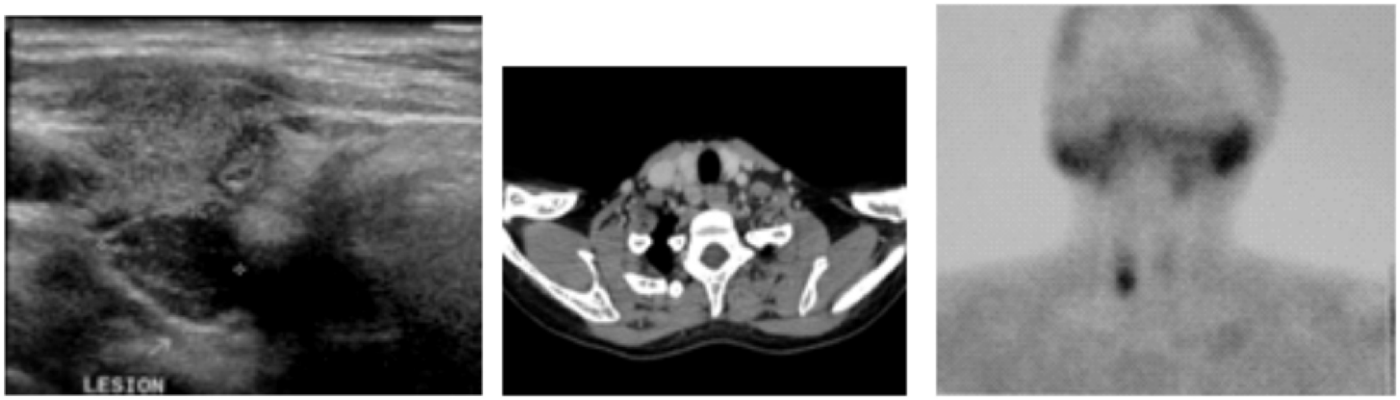


Figure 4: Tc-99m MDP bone scan showing increased uptake of tracer by bones with decreased uptake by soft tissues and kidneys suggestive of superscan.

faint uptake in bilateral kidneys (Figure 4). Consolidating all findings, hyperparathyroidism was suspected and ultrasound neck was performed. There was a well defined elongated hypoechoic lesion just posterior to the right lobe of thyroid (Figure 5A). Computed Tomography (CT) confirmed ultrasound findings with mildly enhancing lesion in the expected location of the right parathyroid gland (Figure 5B). There were associated multiple lytic lesions in ribs and mandible. Further findings were confirmed on Sestamibi Pertechnetate Subtraction Imaging, which revealed a lesion in right parathyroid location (Figure 5C).

DISCUSSION

The bony changes of primary and secondary hyper-parathyroidism are identical. The classical radiographic features of hyperparathyroidism are subperiosteal cortical bone erosions, generalized osteopenia, bone softening, local destructive bone lesions (brown tumors), and calcification of the soft tissue. Pathological fractures may occur, but are infrequent. Subperiosteal bone resorption is most commonly seen involving the hands and the feet, classically along the radial aspects of the second and third middle phalanges. Changes can also be seen involving the



(A) **(B)** **(C)**

Figure 5: Parathyroid adenoma. Well defined parathyroid adenoma is seen posterior to right lobe of thyroid (A,B), Parathyroid adenoma localized on Sestamibi Pertechnetate subtraction scan (C).

medial aspects of the metaphyses of long bones as well as the ribs and lamina dura of teeth and terminal phalyngeal tufts.[2] Chondro-calcinosis is a nonspecific finding that has a higher prevalence in primary rather than secondary hyper-parathyroidism.[3]

Osteosclerosis in primary hyperparathyroidism is rare. It is more commonly associated with secondary hyperparathyroidism. Soft tissue calcification is also commoner in secondary hyperparathyroidism. However, joint cartilage calcification is a common finding in primary hyperparathyroidism.[4]

As observed in this case, spinal involvement presents as striped appearance due to bony osteopenia and end plate sclerosis, the so-called "rugger jersey" spine. Radiographically lytic lesions of brown tumors are more prevalent in primary hyperparathyroidism, but due to increased prevalence of secondary hyperparathyroidism, brown tumors are more frequently encountered in this setting.[4]

On ultrasound, a parathyroid adenoma appears as a hypoechoic solid mass with echogenicity less than that of thyroid gland. About 2% have cystic changes due to degeneration and rarely, they may contain specks of calcification. On CT scan, adenomas are seen as hypo-attenuating masses with contrast enhancement in 20% cases. CT has the added advantage of visualization of bony changes including osteopenia and localization of brown tumors.[3] The present case also showed similar ultrasound and CT findings in adenoma.

MRI due to superiority in picking up marrow signal alteration helps in diagnosing metabolic marrow changes, although differentiation among multiple causes of marrow changes is difficult. MRI is helpful in characterization of brown tumors and differentiation from giant cell tumors, due to presence of products of blood degradation. Although directed ultrasound examination, CT of the neck, and magnetic resonance imaging are useful in the complete evaluation of primary hyperparathyroidism, 99mTc-sestamibi parathyroid scintigraphy is now considered the best preoperative localizing modality for the detection of parathyroid adenomas. As parathyroid scintigraphy can be limited by the coexistence of thyroid nodules or other metabolically active tissues such as lymph nodes, diffuse hyperplasia, or metastatic thyroid cancer, it is often correlated with CT results to yield functional and anatomic localization.[5] Usually for primary hyperparathyroidism, surgery is treatment of choice.[6] NIH has laid down specific guidelines for selection of patients for parathyroidectomy. Few higher centers also perform minimally invasive parathyroidectomy with intraoperative nuclear mapping, also called minimally invasive radio guided parathyroidectomy (MIRP).[3]

CONCLUSION

Hyperparathyroidism is like a pandora box, and need clinical and variety of radiological investigations including radiographs, ultrasonography, or sometimes CT, MRI or nuclear imaging to reach a clinico-radiological diagnosis.

REFERENCES

1. McDonald DK, Parman L, Speights Jr VO. Primary hyperparathyroidism due to parathyroid adenoma. *Radiographics*. 2005;25(3):829-34.
2. Genant HK, Heck LL, Lanzl LH, Rossmann K, Vander Horst J, Paloyan E. Primary hyperparathyroidism: A comprehensive study of clinical, biochemical and radiographic manifestations. *Radiology*. 1973; 109(3):513-24.
3. Sharma P, Parikh K, Chhabra AP, Kapadia B, Vorha PA, Verma S. Classical case of primary hyperparathyroidism. *Indian Journal of Radiology and Imaging*. 2005;15(4): 485-487.
4. Goswami P, Sarma PK, Sethi S, Hazarika S. Extensive skeletal manifestations in a case of primary hyperparathyroidism. *Indian Journal of Radiology and Imaging*. 2002; 12(2):267-70.
5. Johnson NA, Tublin ME, Ogilvie JB. Parathyroid imaging: technique and role in the preoperative evaluation of primary hyperparathyroidism. *American Journal of Roentgenology*. 2007;188(6):1706-15.
6. Eigelberger MS, Cheah WK, Ituarte PH, Streja L, Duh QY, Clark OH. The NIH criteria for parathyroidectomy in asymptomatic primary hyperparathyroidism: Are they too limited? *Annals of Surgery*. 2004; 239(4):528-35.

Corresponding Author

Dr Geetika Sindhwani**,** House Number 12/ Pocket E1, Sector 7, Rohini, Delhi 110085.

email: sindhwani.geetika@gmail.com