A CASE OF PARATHYROID ADENOMA PRESENTING AS PATHOLOGICAL FRACTURES IN YOUNG MALE

KARTHICK KUPPAN
Department of General Medicine,
MADURAI MEDICAL COLLEGE AND HOSPITAL

Abstract:
Primary hyperparathyroidism is a well recognized entity identified almost more than century ago by Van Recklinghausen. This condition is more common in females with peak incidence between 30 to 50 years. Primary hyperparathyroidism is usually diagnosed as an incidental finding of hypercalcemia. Overt bone disease is an extremely rare presentation. Fractures in primary hyperparathyroidism are unusual and usually affects vertebra. We report a case of primary hyperparathyroidism in a young male presented with multiple pathological fractures of long bones without osteolytic lesions.

Keyword: Primary hyperparathyroidism, Pathological fractures

Case Report.
A 17 year old male presented with h/o chronic fatigue, generalized weakness, h/o difficulty in walking and pain in the right thigh after a trivial fall at home. No h/o dyspnoea on exertion/loss of weight/loss of appetite. No h/o polyuria, renal colic, hematuria, acid peptic disorder. No h/o chronic bony pain. Not a known case of DM/Hypertension, CKD. No similar illness in other family members.

General Examination
Patient is conscious & oriented, Ill built & ill nourished, No pallor, No icterus, No cyanosis, No clubbing, No pedal oedema, No lymphadenopathy.

Vitals: Pulse Rate: 88/minute, B.P : 110/70mmof Hg

There were gross appendicular skeletal deformity - cubitus varus, genu valgus, shortening of right leg. Movements were restricted around right hip joint.
Image showing Shortening of right lower limb
Systemic Examination: Motor system: weakness in the proximal girdle muscles bilaterally. Tone was normal, Deep tendon reflexes were preserved. Other systemic examination findings were unremarkable.

Investigations
Hemoglobin - 10.5 g, Total Count - 5200, Differential Count – P60 L35 E2 M3, RBC - 4.1 lakhs, Platelet Count – 2.54 Lakhs, ESR - 25mm/1st hour, Peripheral Smear – hypochromic microcytic anaemia RBS – 84 mg/dl, B. urea - 24 mg/dl, S. creatinine - 0.8 mg/dl, S. Sodium - 139 meq/dl, S. Potassium - 4.4 meq/dl, Routine urine - normal study.

Liver Function Test: Bilirubin: 0.8, SGPT: 38, SGOT: 42, ALP: 659 Total protein: 7 gms
S. Calcium: 13 gms/dl S. Phosphorus: 2.6 gms/dl

Radiological examination:
revealed diffuse osteopenia, thinning of cortex, multiple pathological fractures, segmental fracture of right femur, no osteolytic lesions, no soft tissue calcification. s/o osteogenesis imperfect/hyperparathyroidism.

X Ray Right thigh showing segmental fracture of femur
X Ray Chest PA View showing rib fracture, clavicle and scapular erosion
We proceeded with Parathyroid hormone assay. S. PTH level: 1359 IU/L (60-170 IU/L)
TSH: 2.5 micro IU/L
USG Neck: 2 * 1.7 cm Hypo echoic lesion noted in the left lobe of thyroid. USG Abdomen: cholelithiasis, no renal stones.

CT Neck:

2*1.5 cm hypodense lesion in the left inferior pole of thyroid gland. Secondary bony changes due to hyperparathyroidism was noted

Impression: Solitary left inferior parathyroid adenoma.
A final diagnosis of Primary Hyperparathyroidism due to solitary parathyroid adenoma presenting with multiple pathological fractures was made. Patient underwent parathyroid adenectomy under the care of general surgeon in our institution.
Histopathological examination confirmed the diagnosis of parathyroid adenoma. Following surgery patient developed hypocalcemic...
tetany and treated with Calcium gluconate infusion followed by oral calcium and vitamin D supplements. During follow-up S. Calcium and PTH returned to normal.

Discussion:
Primary hyperparathyroidism is a generalized disorder of calcium, phosphate, and bone metabolism due to an increased secretion of PTH. The elevation of circulating hormone usually leads to hypercalcemia and hypophosphatemia. There is great variation in the manifestations. Patients may present with multiple signs and symptoms, including recurrent nephrolithiasis, peptic ulcers, mental changes, and, less frequently, extensive bone resorption. The manifestations may be subtle, and the disease may have a benign course for many years or a lifetime. The annual incidence of the disease is calculated to be as high as 0.2% in patients >60. The disease has a peak incidence between the third and fifth decades (1). Hyperparathyroidism can be primary, secondary, or tertiary. Primary hyperparathyroidism is of unknown origin and has a sporadic prevalence of 0.2% among women over the age of 40 years and 0.05% among men (4). Eighty percent of patients with primary hyperparathyroidism have a solitary adenoma. Primary hyperparathyroidism is common in females than males in the ratio 3:1. Multiple adenomas occur in 3%–5% of cases, and parathyroid hyperplasia occurs in 15%.. It can also form part of multiple endocrine neoplasia (MEN) syndrome along with medullary carcinoma of thyroid, phaeochromocytoma, and mucosal/cutaneous neurofibromata or pituitary adenoma and islet cell tumours of the pancreas.(2) Half or more of patients with hyperparathyroidism are asymptomatic. Manifestations of hyperparathyroidism involve primarily the kidneys and the skeletal system. Kidney involvement, due either to deposition of calcium in the renal parenchyma or to recurrent nephrolithiasis.

The distinctive bone manifestation of hyperparathyroidism is osteitis fibrosa cystica, which occurred in 10–25% of patients in series reported 50 years ago. Histologically, the pathognomonic features are an increase in the giant multinucleated osteoclasts in scalloped areas on the surface of the bone (Howship's lacunae) and a replacement of the normal cellular and marrow elements by fibrous tissue. Bone disease is rarely overt. Radiographic manifestations are seen in less than 2% of patients and include subperiosteal erosions, diffuse osteoporosis, cystic lesions (‘brown tumours’), pathological fractures, ‘salt and pepper’ mottling of skull and loss of lamina dura in the mandible(1).

Two distinct types of bone lesion in PHPT are described one, the rapidly progressive type, classical osteitis fibrosa cystica is seen. The second, slowly progressive, leads to cortical thinning and osteoporosis.11 Pathological fractures may occur through a cyst or in weakened long bones.(2)

The causes of pathological fracture include(3)
1. Primary bone disorders like Osteogenesis Imperfecta, Pagets disease, Osteoporosis, Simple bone cyst, Aneurysmal bone cyst.
2. Endocrine Disorders like hyperparathyroidism, hyperthyroidism.
3. Neoplasms and bony metastasis.
4. Infections

The incidence of pathological fractures in primary hyperparathyroidism is quite low and apart from vertebral fractures no characteristic pattern have been described (4). The prevalence of bone disease is much greater in patients with parathyroid carcinoma than it is in patients with parathyroid adenoma with
70% or fewer patients manifesting symptoms related to calcium absorption with osteoporosis and bone pain. Parathyroid cancers are hyperfunctional unlike other endocrine tumors that become less hormonally active when malignant.(5)

Extensive bony involvement with pathological fractures as presenting feature due to parathyroid carcinoma has documented but multiple pathological fractures as presenting feature of primary hyperparathyroidism due to parathyroid adenoma is extremely rare.(4)

References:
1. Harrison’s Principles of Internal Medicine 17th Edition Pages:2377 to 2383


