

Short Report

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Osteitis fibrosa cystica in primary hyperparathyroidism**Suhas Sondur***; Aditya Prasad Panda

Department of Orthopaedics, Kalinga Institute of Medical Sciences, KIIT University, Bhubaneswar, Odisha, India.

***Corresponding Author: Suhas Sondur**

Department of Orthopaedics, Kalinga Institute of Medical Sciences, KIIT University, Bhubaneswar, Odisha, India.

Tel: +91-7259959943;

Email: sondur.suhas@gmail.com

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Image description

A 29-year-old woman with no known medical history presented with a one-year-old pathological fracture of her left femur following a trivial fall at her residence. Initially treated with an intramedullary nail, she complained of difficulty in walking and multiple bony aches. Physical examination revealed cachexia, multiple bony tender points, and bony swelling over her parietal bone. Sequential x-rays of her femur revealed thinning of cortices, generalized osteopenia, and scalloping of the medial femoral cortex. The latest radiograph (Figure 1, Panel A) showed diffuse cystic enlargement of the midshaft femur with multiple septae, a thinned-out cortex, diffuse osteopenia, and a loosened implant. A skeletal survey was performed which revealed the following features (Figure 1): Panel B: Thinning of metacarpal head cortex with a cystic enlargement (yellow arrow) classically named "*osteitis fibrosa cystica/Browns tumor*"; Panel C: skull radiograph with a cystic enlargement of the parietal bone with sclerotic margins and generalized osteopenia; Panel D: Multiple calcifications (yellow arrow) in both kidneys (nephrocalcinosis). Ultrasonography of the abdomen showed chronic calculous cholecystitis and bilateral medullary nephrocalcinosis. Biochemistry was notable as serum calcium was 14.1 mg/dl [reference 8.6 to 10.3], serum phosphorous was 1.8

mg/dl [reference 2.5 to 4.5], serum parathyroid hormone was 1004.4 pg/ml [reference 15 to 65], serum Alkaline phosphatase was 497 U/L [reference 35 to 104] and the 24 h urinary calcium was 455 mg [reference 100 to 300]. A diagnosis of primary hyperparathyroidism was made, confirmed by a right-sided parathyroid adenoma measuring around 0.8 x 0.8 x 0.6 cm on ultrasound of the neck and Tc-99m Sestamibi scan (Figure 2).

Discussion

Primary Hyperthyroidism (PHPT) is characterized by hypercalcemia and increased Parathyroid Hormone (PTH) levels in the bone. Parathyroid adenoma comprises 90% of all primary hyperparathyroidism patients and the remaining parathyroid hyperplasia [1]. PTH directly stimulates osteoclasts directly through its receptors and RANK-L and M-CSF synthesis and stimulates bone resorption. Excessive PTH levels lead to increased bone resorption from cortical bone and consequently increased calcium levels. This leads to areas of focal excessive bone resorption leading to cyst formation and replacement with fibrous tissue classically called osteitis fibrosa cystica or Brown tumor of the bone [2]. The other skeletal features include subperiosteal resorption of bone, tapering of small bones, and salt and pepper appearance of the skull [2]. Other features include

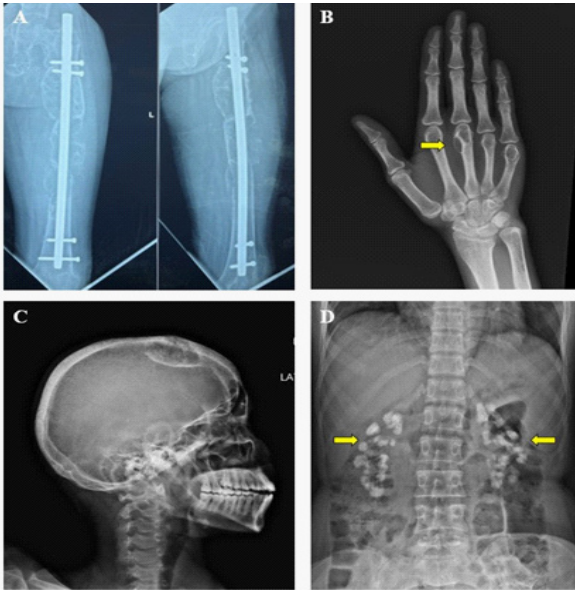


Figure 1:

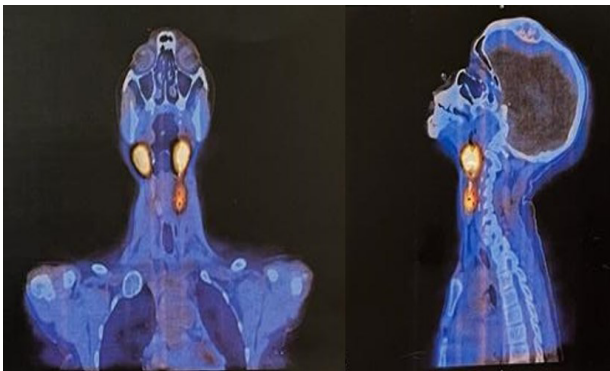


Figure 2:

nephrocalcinosis, nephrolithiasis, constipation, muscle weakness, and hypertension [3]. Hence the classical description is “the disease of stones, bones, and groans”. Laboratory tests in PHPT denote increased serum calcium levels, increased PTH levels and reduced serum phosphorous levels, and increased urinary calcium. Along with the radiography, the Technetium-99 Sestamibi scan is specific to diagnose parathyroid adenoma/hyperplasia [4]. Treatment of PHPT is by complete or partial parathyroidectomy and a successful surgery results in permanent normalization of calcium levels and improvement of bone mineral density [5].

Declarations

Conflicts of interest: Nil.

Research involving human participants and/or animals: Nil.

Informed consent: Informed consent was taken from the patient for this publication.

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