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Classical Imaging Findings in Osteomalacia Due to Nutritional Deficiency.

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ABSTRACT

21 years female presented with complaints of inability to walk with gradual weakness and diffuse bone pain for 3 years lab investigations and radiological survey, ultrasound, CT and nuclear scans revealed multiple symmetrical looser zones representing osteomalacia due to nutritional deficiency. Since full blown nutritional deficient osteomalacia with typical features is very rare in current scenario we would like to present this case re-emphasizing florid osteomalacia skeletal manifestations.

Keywords: osteomalacia, nutrition, deficient, multiple, looser zones

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INTRODUCTION

Vitamin D deficiency in adult bone results in osteomalacia .In mature skeleton with fusion of growth plates , pathognomonic radiographic feature is looser' s zone or pseudo fractures/ milkman's fractures. This young female typically presented with this feature and various imaging modalities like multiple radiographs, ultrasound neck, CT neck and technetium 99m Sestamibi scan for parathyroid were done that showed various findings substantiating the diagnosis

Case Report

Female aged 21 years presented with complaints of inability to walk since 6 months. Patient was apparently normal 3 yrs before. To start with she had pain and difficulty in walking with associated hip instability so she walked with support. Since six months the patient stopped walking even with support. She has diffuse bone pain. No relevant past / family history

Lab investigations of serum calcium was normal (9.1 mg /dl).Serum ALP was elevated (629 lu/l), Vitamin D3 was low (6.8 ng/ml),serum parathormone was elevated (576 pg.). Serum urea, creatinine, phosphorous were normal.

Multiple radiographs for skeletal survey was done. Generalized osteopenia was noted xray skull (Fig 1) appeared normal .X ray hands (Fig 1)showed intra cortical tunneling . X-ray cervical (Fig 2)and dorsal spine(Fig 3) showed C2-C6 spinous process , axillary borders of both scapulae looser zones with left clavicle anterior cortex erosion, rugger jersy vertebrae, rib fractures, x-ray lumbar spine (Fig 4)and pelvis(Fig 5)demonstrated deformed triradiate pelvis with multiple pseudo fractures involving pelvic bones with acute angulation at S2-S3 levels .Knee radiographs(Fig 6) showed symmetrical looser's zones in proximal fibula, widening and delayed epiphysis fusion of femur. Ultrasound neck showed no parathyroid region SOL. USG abdomen and pelvis revealed normalstudy.

CT neck (Fig 7)demonstrated thymic hyperplasia with c2-c6 spinous fractures, CT chest, abdomen and pelvis (Fig 8a,b)illustrated multiple symmetrical rib fractures, fracture lateral aspect of axilla, rugger jersy spine, deformed triradiate pelvis with multiple pseudo fractures in pelvic bones, altered contour of left femoral neck and acute angulation at S2/S3. Technetium 99m Sestamibi scan (Fig 9)was done to look for parathyroid adenoma which showed a focal area of hyperactivity which was secondary to hyperparathyroidism

Figure 1: X-ray skull - normal .X ray hands - intra cortical tunneling.



Figure 2: X-ray cervical spine- C2-C6 spinous process, axillary borders of both scapulae looser zones with left clavicle anterior cortex erosion



Figure 3: X-ray d spine-multiple symmetrical looser zones in ribs, ruggerjersy spine



Figure 4: X-ray lumbar spine shows deformed triradiate pelvis with multiple pseudo fractures involving pelvic bones with acute angulation at S2-S3 levels.



Figure 5: X-ray ankle—widening of epiphysis. Xray pelvis- Protrusio acetabuli, triradiate pelvis with pelvic bones pseudo fractures



Figure 6: Knee radiographs showed symmetrical looser’s zones in proximal fibula, widening and delayed epiphysis fusion of femur



Figure 7: CT neck with 3 D reconstruction shows c2-c6 spinous fractures,

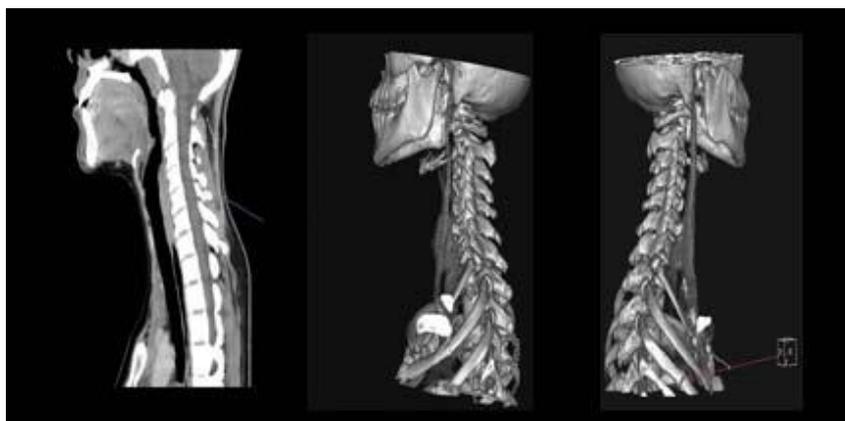


Figure 8a, b: CT chest, abdomen and pelvis - symmetrical rib fractures, fracture lateral aspect of axilla, rugger jersy spine

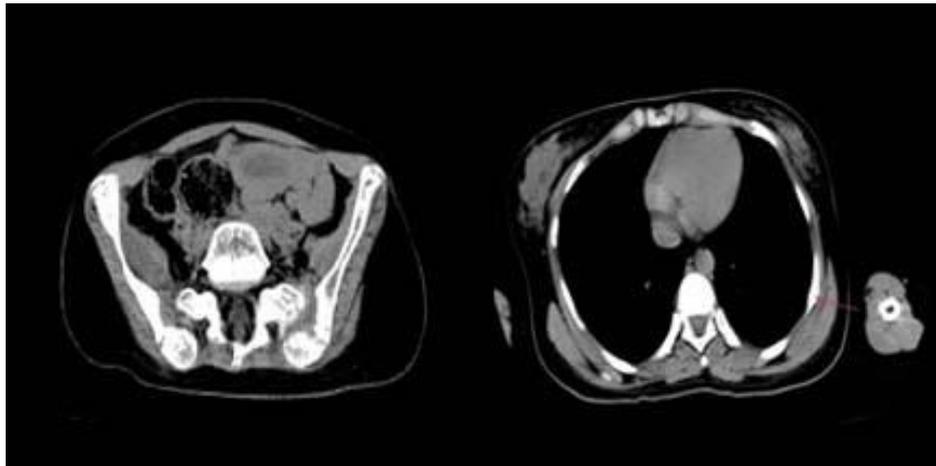


Figure 8b: Deformed triradiate pelvis with multiple pseudo fractures in pelvic bones

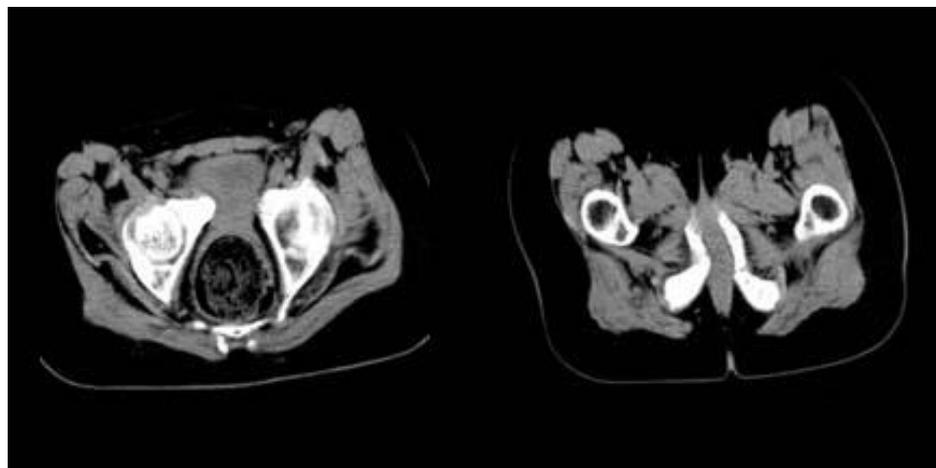
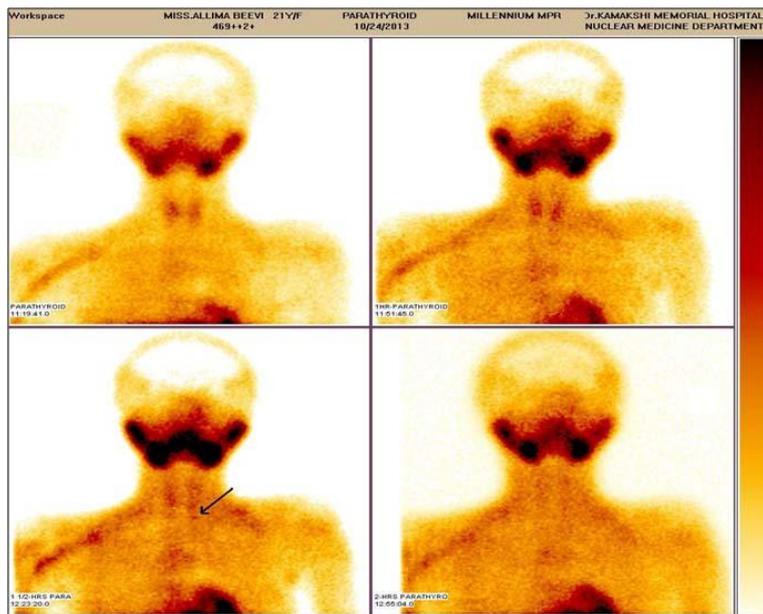


Figure 9: Technetium 99m Sestamibi scan -focal area of hyperactivity



DISCUSSION

Nutritional vitamin d deficiency results in defective mineralization of osteoid leading to qualitative abnormality of skeletally mature bone [1]. mineralization of bone matrix depends on presence of adequate 1,25 –dihydroxy vitamin D ,calcium, phosphorus and alkaline phosphatase, and normal body ph. Deficiency leads to systemic acidosis, mineralization of bones becomes defective leading to qualitative abnormality of bone-reduction in mineral to osteoid ratio leading to rickets in immature bone and osteomalacia in mature skeleton in adults. Etiologies of osteomalacia includes vitamin D deficiency, lack of sunlight exposure [2], malabsorption states like small bowel diseases, Chronic liver disease, Chronicrenal disease, X-Linked hypophosphataemia-phosphaturia, hypophosphatemia, tumor induced oncogenic osteomalacia, phosphaturia, hypophosphatemia (phosphatonin) as in haemangiopericytoma, skeletal tumors and neuro fibromatosis, long term anticonvulsant therapy. Any process preventing normal supply of calcium being absorbed by the intestine leads to osteomalacia [3]. VitaminD aids in absorption of calcium and hence deficiency of vitamin d leads to osteomalacia. Clinical features are nearly specific bone pain and muscle weakness and wasting without neurological damage [5].

RADIOLOGICAL FINDINGS

At skeletal maturity enchondral ossification process ceases leading to defective mineralization of osteoid evident radiographically as looser's zones pathognomonic of osteomalacia [1]. Looser zones are pseudo fractures that has unmineralised osteoid which are typically bilateral and symmetrical .Radiographhically they appear as radiolucent lines perpendicular to bone cortex and does not involve entire bone cortex typically in the medial aspect of femoral neck, pubic rami,lateral border of scapula and ribs involving first two ribs where traumatic fractures are uncommon [1]. In late stages the shape of pelvis also becomes deformed giving characteristic triradiate pelvis [2]. By definition, osteomalacia occurs only in adults and thus has no effect on the growth cartilage [4]. In adults with osteomalacia, theosteopenic bone changes may dominate the picture [4]. In osteomalacia hypocalcaemia acts as a stimulus to secondary hyperparathyroidism. Radiological features of secondary hyperparathyroidism includes bone softening and deformity – Protrusio acetabuli, triradiate pelvis, bowing of long bones ,sub periosteal resorption noted at phalanges , SI joints, symphysis pubis, proximal tibia, outer end of the clavicle and skull(pepper pot skull) ,cortical tunneling, hazy trabecular pattern ,generalized osteoporosis with biconcave end plates due to deformation of malacic bone by the intervertebral disc leading to cod fish vertebral deformity.

Osteomalacia with associated renal osteodystrophy shows osteomalacia features with secondary hyperparathyroidism (erosions, osteosclerosis, and brown tumors), soft tissue and extensive vascular metastatic calcification and adynamic bone development.New complications includes amyloid depositonn, non-infective spondyloarthropathy, osteonecrosis and calciphylaxis-soft tissue calcifications with ischemic necrosis f skin, muscle and subcutaneous fat.

Differential Diagnosis

Secondary to primary hyperparathyroidism is ruled out due to normal serum parathormone levels following treatment with high vitamin d doses and normal sestambi scan findings, Secondary to renal osteodystrophy is ruled out as the renal parameters were normal. Looser's zone Vs.insufficiency fractures - Multiple micro fractures in brittle osteoporotic bones showing florid callus formation suggestive of insufficiency fracture, Incremental fracture in paget's disease resembles looser's zone

CONCLUSION

Vitamin D deficiency in adult bone leads to osteomalacia which can result in multiple bone fractures and deformities with bone pain increasing morbidity to the patient .Early diagnosis and treatment helps preventing permanent deformities.This case re -emphasizes florid osteomalacia skeletal manifestations.

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