

# Image of Interest Secondary Hyperparathyroidism



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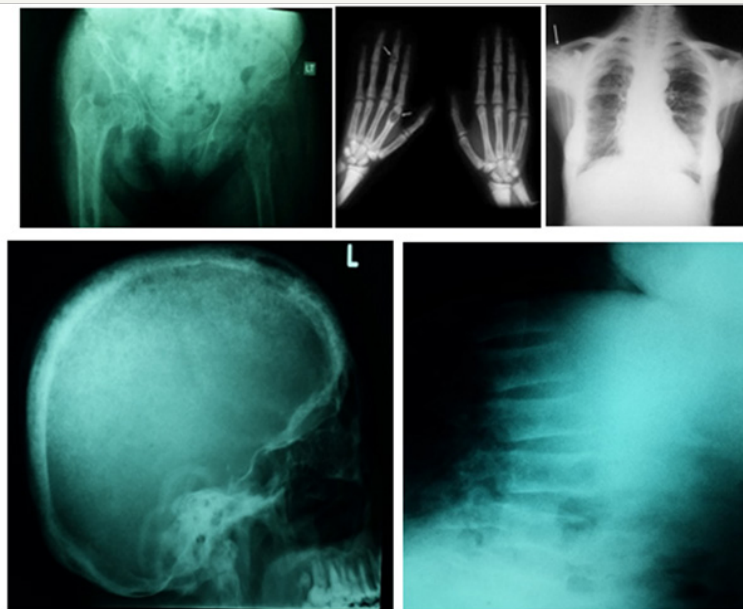
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## Case Report

35 year old lady with CKD on haemodialysis for 10 years was admitted with left hip pain after a trivial trauma. Plain radiograph

of pelvis including both hips was advised. Based on the pelvic radiographic finding further radiographs of hand, shoulder, skull, and spine were obtained (Figure 1).



**Figure 1:** Pelvic radiographic finding further radiographs of hand, shoulder, skull, and spine were obtained.

### What is the diagnosis?

a. Secondary hyperparathyroidism: Fracture in this case was the result of relatively trivial trauma, raising suspicion of cortical thinning, difficult to manage by ORIF due to poor bone quality. We applied skin traction with close monitoring to avoid bed ridden complication and manage fracture. Work up for secondary hyperparathyroidism confirm by serum parathyroid hormone (PTH > 389.9 pmol/L), calcium (1.91 mmol/L), and phosphorus level (1.55 mmol/L). Secondary hyperparathyroidism is characterized by pronounced parathyroid gland hyperplasia resulting from end-organ resistance to parathyroid hormone (PTH). The consequent hyper secretion of PTH depresses calcium levels [1]. The most important cause of secondary hyperparathyroidism is chronic renal insufficiency. The clinical manifestation of secondary

hyperparathyroidism includes bone and joint pain, as well as limb deformities.

b. Pathology: Increased levels of parathyroid hormone (PTH) lead to increased osteoclastic activity. The resultant bone resorption produces cortical thinning (subperiosteal resorption) and osteopaenia [2].

c. Preferred examination: Radiographs are the main stays diagnosis of secondary hyperparathyroidism, because the predominant changes are skeletal, with abnormal calcifications at various sites; these calcifications are well depicted on conventional radiographs [2].

### What is the typical radiological finding?

a. List of typical radiological findings: Lacativ et al. [3] was obtained from 73 chronic hemodialysis patients with

severe HPT2. The regions of radiographic studied were the skull, hands, wrists, clavicles, thoracic and lumbar column, long bones and pelvis. The most common abnormality was subperiosteal bone resorption, mostly at the phalanges and distal clavicles 94%, 'Rugger jersey spine' sign was found in 27%. Pathological fractures and deformities were seen in 27% and 33%, respectively. Calcifications were presented in 80%. Brown tumors were present in 37%.

b. Subperiosteal resorption: Classically at the distal phalangeal tufts (acroosteolysis) and along the radial margins of the second and third middle phalanges.

c. Trabecular resorption: Resorption within medullary bone gives bone a granular appearance, with loss of distinct trabecular detail. In the skull, the diploic space is replaced by connective tissue, leading to a speckled appearance ("salt and pepper" skull).

d. Brown tumors: Accumulations of osteoclasts and fibrous tissue. Tend to heal after treatment of the underlying disorder. Eccentric/intracortical, lytic and often expansile. Incidence is greater in primary hyperparathyroidism, but more commonly seen with secondary hyperparathyroidism due to the higher prevalence [3,4].

e. Soft tissue calcifications: More commonly seen in secondary Hyperparathyroidism.

f. Bone sclerosis: More commonly seen in secondary hyperparathyroidism. Can be seen in the metaphyses of long bones, the skull, or the vertebral body endplates (rugged jersey spine). Progressive hypertrophy of the facial and cranial bones can produce "leontiasis ossea" (lion face), and can mimic Paget disease and fibrous dysplasia [4].

## References

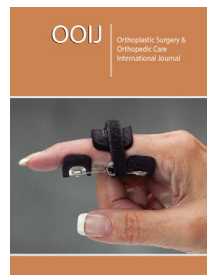
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