Pathological Fractures as the Presenting Symptom of Parathyroid Adenoma: A case Report

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Abstract: Primary Hyperparathyroidism is usually diagnosed as an incidental finding of hypercalcemia in blood tests or due to symptoms secondary to the high calcium. Overt bone disease is an extremely rare presentation. Fractures in hyperparathyroidism are unusual and usually affect the vertebrae. Generally pathological fractures have been described in patients with parathyroid carcinoma. We report a case with benign parathyroid adenoma who primarily presented with pathological fractures of long bones. Excision of the parathyroid adenoma brought the normal biochemistry profile and increases the bone density, this leads to reunion of fracture.

I. Introduction

Primary hyperparathyroidism is usually diagnosed as a result of chance finding of raised serum calcium or complications associated with hypercalcemia such as polyuria, polydipsia, muscle weakness, gastrointestinal upsets and renal stone formation. 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. Incidence of fractures in hyperparathyroidism is quite low and about 10% in two large series and apart from vertebral compression fractures, no characteristic fracture pattern have been described.

Pathological fractures with extensive bony involvement are presenting features of parathyroid carcinoma literature, but multiple pathological fractures, as a presenting feature of primary hyperparathyroidism due to parathyroid adenoma is extremely rare.

Problems and difficulties with parathyroid surgery are pronounced in younger patients. The high rate of multiple gland disease requires bilateral cervical exploration as the standard procedure in PHPT patients younger than 30 years of age.

II. Case report

A 15-year-old female presented to emergency after tripping on a step and falling onto her right side. She was otherwise fit and well. Radiographs confirmed a sub-trochantric fracture of femur through an area of lytic bone. Plain radiographies of the hands, skull, clavicle, and sacroiliac joints were performed but no abnormalities were found. The patient bone profile revealed markedly raised calcium of 3.3 mmol/l (corrected range: 2.15–2.60) and urinary calcium 506 mg/day (100–300). The creatinine level was within normal limits. A PTH assay objectified a circulating level of PTH of 160 pg/mL (13–54) and the diagnosis of PHPT was then confirmed. A cervical ultrasound identified an adenoma in the left inferior parathyroid gland. Tc-99 sestamibi scan confirmed increased uptake in left inferior parathyroid gland and no other area of increased uptake was detected. A parathyroidectomy was performed and a large (13 g) parathyroid was removed and confirmed to be a benign parathyroid adenoma on histological examination. Despite a transient postoperative hypocalcaemia, the patient made an uneventful recovery and discharged. Two weeks later, the level of PTH was normal (48 pg/mL).
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Figure 1. CECT neck showing left inferior parathyroid adenoma (black arrow)

Figure 2. Tc-99 sestamibi parathyroid scan showing increased uptake in left inferior parathyroid gland (black arrow) and uptake is still there after two hours (white arrow)

Figure 1. Pathological fracture
The incidence of PHPT is quoted at 2–10/10,000 of the population. Of these, 50–70% are now routinely detected on incidental biochemical assays. Up to one third of patients may be asymptomatic. Bone pain and tenderness are seen in PHPT but occur more commonly in secondary hyperparathyroidism. Fracture occurs very rarely in HPT. The crude fracture rate in patients known to have PHPT has been documented at 15/1000 person years compared to 8/1000 in controls. However, PHPT revealed by pathological fracture, as in our case, is very uncommon.

Our patient had a history of falling, and considering her age, osteoporosis was probably associated to some extent. Interestingly, no correlation had been identified between fracture risk and preoperative calcium levels or the weight of the diseased parathyroid removed at surgery. These fractures are due to brown tumor which is a rare complication of PHPT. Brown tumors are benign focal bone lesions caused by increased osteoclastic activity and fibroblastic proliferation, encountered in primary or more rarely secondary hyperparathyroidism. These tumors may appear in any bone but are frequently found in the facial bones and jaws, sternum, pelvis, ribs, femur, and rarely the vertebrae. The histological findings of the tumor are similar to those of giant cell tumors and aneurismal bone cyst and may cause confusion in diagnosis. Nevertheless, because of the difficulty of microscopic differential diagnosis, the clinical presentation and the biochemical findings should be considered carefully for making the correct diagnosis. The increased PTH value is the determinant in diagnosis. In X-rays, brown tumors are seen as lytic lesions with regular borders. The cortex can be seen as narrowed and extended; however, there is no penetration. These large bone defects increase the spontaneous fracture risk. Our patient's radiologic views were also consistent with these findings. CT is useful for evaluating brown tumors of lower limbs to show their uniform tissue density and the contrast enhancement of the lesion. However, when CT shows a fluid-filled cyst, local surgery must be performed to prevent a pathological fracture. Brown tumors are hypervascular and therefore enhance on MRI, and intensely active in bone scintigraphy. In addition to the surgical stabilization of the femur, aggressive treatment of hyperparathyroidism is also necessary to achieve successful outcomes. Thus, the increased risk of fracture disappears within a year following surgery suggesting a quick restoration of bone biomechanical competence.

References