Case Report

# Bone Under Siege: A case of severe Skeletal Disease in Primary Hyperparathyroidism

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## ABSTRACT

Primary hyperparathyroidism can lead to rare but severe bone disease. We report a 28-year-old male with chronic generalized body aches, fatigue, weight loss, difficulty walking, and painful limb movements over several months. Symptoms progressed to multiple pathological fractures of the left femur, right femoral neck, left tibia, left proximal humours, and left proximal radius and ulna, confirmed radio logically. Laboratory evaluation revealed elevated PTH and calcium with low phosphorus and vitamin D. Bone biopsy from the left tibia showed giant cell lesions consistent with osteitis fibrosa cystica. Ultrasound confirmed a parathyroid adenoma, which was surgically removed along with partial thyroidectomy. Postoperative follow-up showed normalized PTH, calcium, and vitamin D levels with complete symptom resolution. This case highlights the importance of early detection and surgical management in primary hyperparathyroidism.

**KEY WORDS:** Hyperparathyroidism; Bone Fractures; Osteitis Fibrosa Cystica; Parathyroid Neoplasms; Para thyroidectomy.

## INTRODUCTION

Primary hyperparathyroidism is a disease of the thyroid gland leading to increased secretion of parathyroid Harmon (PTH) either due to parathyroid adenoma, parathyroid hyperplasia or carcinoma of the parathyroid gland.[1] Parathyroid glands are four in numbers usually located posteriorly to thyroid gland. Parathyroid adenoma is the most common cause of hyperparathyroidism and contributes 85-90% as a cause of primary hyperparathyroidism with 10-15% being double adenoma or multi-gland hyperplasia.[2-4] The increase secretion of parathyroid hormone leads to increase bone reabsorption which leads to hyperkalaemia and hypophosphatemia.[5]

Although primary hyperparathyroidism can be asymptomatic but it can present with gastrointestinal symptoms like constipation, peptic ulcer disease, and acute pancreatitis and increased gastric acid secretion.[6] Lethargy, depression, fatigue and weakness are the neurological and neuropsychiatric complications that may develop during the course of the disease condition. Such patient may also present with dental problem.

Pathological fractures of long bones are the rare but the most concerning feature of primary hyperparathyroidism due to adenoma. Herein we present a case of parathyroid adenoma induced primary hyperparathyroidism leading to pathological fractures of long bones.

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## CASE PRESENTATION

A 28-year-old Afghan male presented to the Orthopaedic Department of Northwest General Hospital, Peshawar, with painful swelling of the left leg after a minor twist. Radiology revealed a pathological fracture of the left tibia. Over the past three years, he experienced generalized body aches, fatigue, difficulty walking, weight loss, and recurrent fractures of multiple long bones, including the femur, tibia, humerus, radius, and ulna, each occurring with minimal trauma. The patient had multiple admissions and treatments in Afghanistan and was often lost to follow-up due to financial constraints; however, there were no other obvious clinical features apart from those related to hyperparathyroidism

Despite multiple presentations to local orthopaedic facilities in Afghanistan, the patient was never comprehensively evaluated for an underlying metabolic or systemic cause. This time, however, the orthopaedic team at Northwest General Hospital took a broader diagnostic approach. Routine blood investigations revealed markedly elevated serum calcium levels, prompting a referral to the Endocrinology Department.

Endocrine evaluation showed significantly elevated parathyroid hormone (PTH) levels at 1349 pg/ml (normal: 15–65 pg/ml), accompanied by hyperkalaemia (13.3 mg/dL), elevated alkaline phosphatase (601 IU/L), low phosphate (1.79 mg/dL), and vitamin D deficiency (13.3 ng/ml). The normal laboratory reference ranges are provided in Table-I for comparison. These findings raised strong suspicion for primary hyperparathyroidism. A neck ultrasound further revealed a left-sided parathyroid adenoma, while abdominal imaging detected renal calculi. The patient was also evaluated clinically for Multiple Endocrine Neoplasia type 1 (MEN-1). No symptoms or family history suggestive of MEN-1 were identified, although genetic testing could not be performed due to financial constraints.

During his hospital stay, the patient was taken to the operating theatre for a bone biopsy from the fractured left tibia, which histologically revealed a giant cell lesion consistent with Osteitis Fibrosa Cystica, a classic skeletal manifestation of severe hyperparathyroidism.

A final diagnosis of Primary Hyperparathyroidism secondary to a parathyroid adenoma was made. The patient subsequently underwent a Para thyroidectomy along with subtotal thyroidectomy. Postoperatively, his PTH and calcium levels normalized (PTH: 25.2 pg/ml, calcium: 9.3 mg/dl). He was initiated on oral calcium and vitamin D supplementation and advised regular follow-up.

On reassessment six months later, his biochemical profile continued to show stability: PTH decrease to 25 pg/ml, calcium remained normal at 9.7 mg/dl, phosphate had normalized to 3 mg/dl, and vitamin D levels had improved significantly to 98 ng/ml. Written consent form was obtain from the patient.

## DISCUSSION

The incident of primary hyperparathyroidism significantly increase with age however it varies in different age groups and predominantly effects female.[7] Peak age of incident is 30-50 years.[8] According to some studies the incident of primary hyperparathyroidism in post-menopausal women is more than 2 present.[9]

Primary hyperparathyroidism is relatively rare in clinical setting and is therefore considering a reason to missed diagnosis due to non-specific symptoms shared with other conditions.[10] Patients with severe bone disease due to primary hyperparathyroidism often present to orthopaedic or bone oncology departments, which may lead to delayed diagnosis and management, or even a missed diagnosis of the underlying condition.[11] Missed or delayed diagnosis can further aggravate the symptoms with grave deterioration of the patients and increasing morbidity due to severe bone condition like osteitis fibrosis cystica, recurrent fractures and delayed fracture

Table-I: Laboratory investigations at three time point.

| *Investigation* | *Normal Range* | *Before Surgery* | *After Surgery* | *6 Months After Surgery* |
|---|---|---|---|---|
| PTH | 15-65pg/ml | 1349 | 25.20 | 25.0 |
| Alkaline Phosphatase | 30-130IU/L | 601 | 400 | 100 |
| Calcium | 8.5-10.5mg/dl | 13.3 | 9.3 | 9.7 |
| Ionized Calcium | 4.56-5.40mg/dl | 8.27 | 5 | 4.8 |
| Phosphorus | 2.6-4.5mg/dl | 1.79 | 2 | 3 |
| Vitamin-D | 30-40ng/ml | 13.3 | -- | 98 |
| TSH | 0.35-4.94mI/ml | 1.02 | -- | 1.35 |
| Testosterone | 4.94-32.01 ng/ml | 5.90 | -- | 15 |

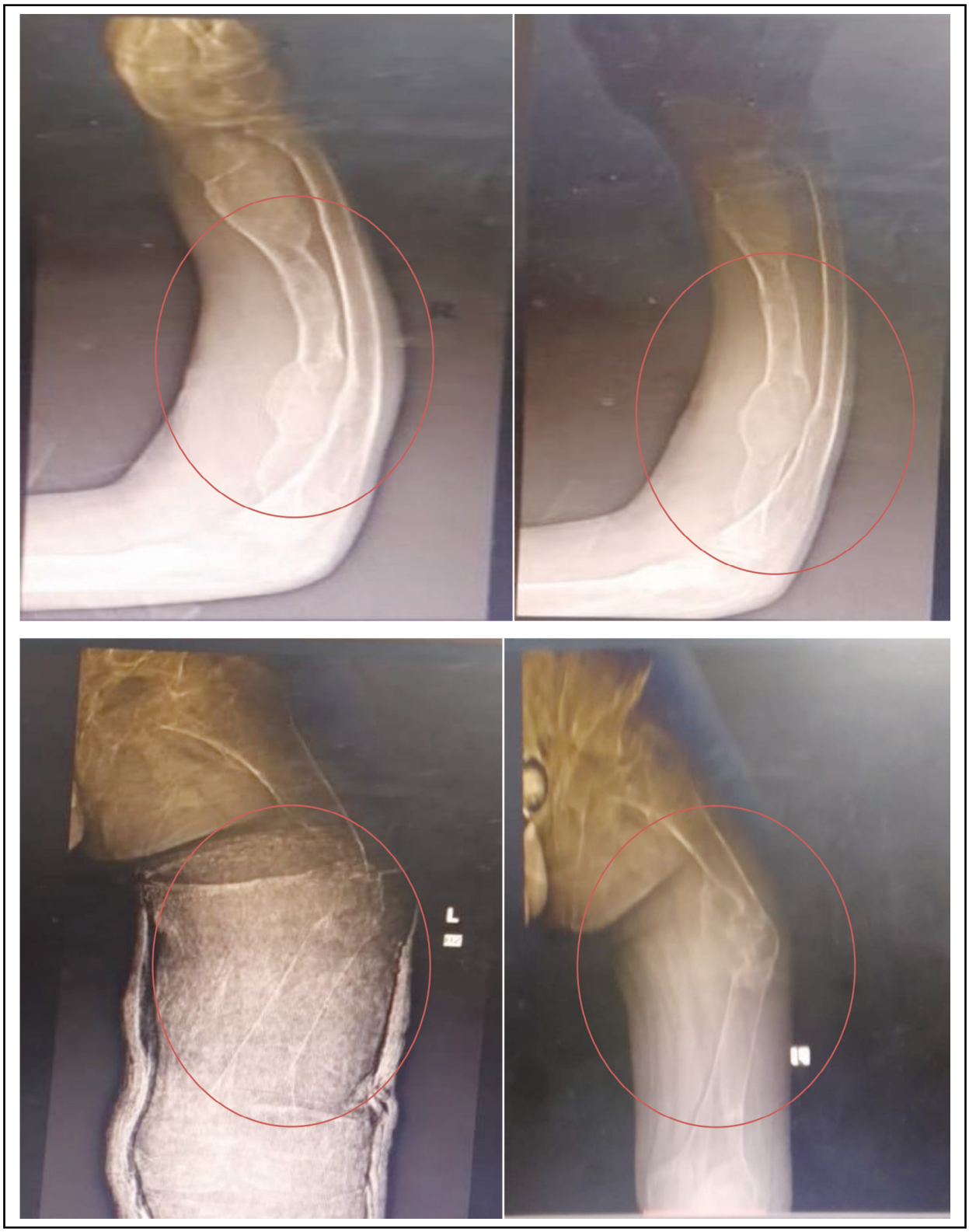

Fig.1: Radiographic views demonstrating fracture displacement and alignment, with Image 4 providing an additional projection for further clarification. Images 1–3 show antero-posterior and lateral radiographic views demonstrating fracture displacement and alignment. Image 4 provides an additional complementary view that further clarifies the fracture configuration.

healing. Once the disease is diagnosed, surgical removal of the parathyroid gland is the treatment of choice which leads to normalization of serum parathyroid Harmon, serum calcium and phosphorous. The vitamin D level also normalises after removal of parathyroid gland.

Primary hyperparathyroidism is also reported in syndrome form in approximately 5 % of patients, most commonly as a part of MEN-1 syndrome. Therefore, it is of prime importance to evaluate for associated features when primary hyperparathyroidism is diagnosed and to pursue further investigations if indicated.[12]

After Para thyroidectomy patient can develop symptoms of hypocalcaemia, which should be monitored clinically and biochemically. Patient may require oral or iv calcium if any symptoms of hypocalcaemia develop. In summary we discussed a rare presentation of recurrent pathological fractures of primary hyperparathyroidism with biochemical evidence of high serum calcium, low phosphorus, high PTH and low vitamin D level. After surgical removal of parathyroid gland, the patients become asymptomatic with normalization of serum PTH, calcium and phosphorous.

## CONCLUSION

A high index of suspicion is necessary to diagnose the unusual and rare presentation of primary hyperparathyroidism. This case report highlights a rare but clinically significant possibility of severe bone disease, presented with history of recurrent pathological fractures of long bones in patient with primary hyperparathyroidism due to parathyroid adenoma. Early recognition and surgical removal of parathyroid adenoma are of prime importance and treatment of choice in such symptomatic cases.

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**IU:** Manuscript writing and ethical clearance.
**AH:** Consultant endocrine of the patient.
**WA:** Support in writing.

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