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CASE SERIES

Skeletal Manifestations of Primary Hyperparathyroidism: A Series of Four Cases

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Abstract

Primary Hyperparathyroidism (PHPT) has undergone a marked epidemiological shift worldwide, with Western countries reporting predominantly asymptomatic, screening-detected disease, whereas India continues to see a substantial burden of symptomatic PHPT. This case series describes four patients presenting with advanced skeletal manifestations of PHPT, highlighting diagnostic challenges, biochemical severity, and surgical outcomes. All patients presented with severe skeletal disease, including pathological fractures, brown tumours, and marked osteoporosis. Mean serum calcium and PTH of the cases were 13.7±1.5 mg/dL (12.3-15.1mg/dl) and 1960±929 pg/mL (870-2956 pg/ml) respectively. Diagnostic delay averaged 11.8 months, as all patients initially underwent extensive orthopaedic or oncologic evaluation. All four underwent parathyroidectomy for solitary adenomas and achieved complete biochemical cure. Hungry Bone Syndrome (HBS) occurred in all cases, requiring intravenous calcium. At one year, all patients demonstrated substantial clinical improvement, normalization of calcium and PTH, marked reduction in alkaline phosphatase, and significant gains in bone mineral density. Despite global declines in symptomatic PHPT, advanced skeletal disease persists in India, often compounded by vitamin D deficiency and diagnostic delay. Parathyroidectomy remains highly effective, producing excellent biochemical cure and dramatic skeletal recovery even in severe disease. Early calcium and PTH testing in patients with lytic lesions or unexplained fractures is essential for timely diagnosis.

Keywords: Brown Tumour, Osteitis Fibrosacystica, Primary Hyperparathyroidism

1. Introduction

Primary Hyperparathyroidism with severe bone manifestations has become increasingly rare in developed countries, with fewer than 5% of patients now presenting with overt skeletal disease due to early detection through routine screening. In developing countries, including India, the clinical profile is rapidly evolving. With greater awareness among physicians, widespread use of autoanalyzers, and increasing trend of vitamin D supplementation, it is expected that the clinical and biochemical presentation of this entity is likely to change. However, severe skeletal manifestations including brown tumour and pathological fractures continue to occur, particularly in rural areas and cases with delayed diagnosis, representing persistent clinical challenges that require recognition and appropriate management. This case series presents four cases diagnosed with primary hyperparathyroidism presenting predominantly with skeletal manifestations, highlighting the clinical features, biochemical profiles, radiological findings, surgical management, and outcomes.

2. Aim and Objectives

- To describe the clinical, biochemical and radiological features of four patients with PHPT presenting with skeletal disease.

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- To assess surgical management and postoperative outcomes.

3. Review of Literature

Primary Hyperparathyroidism (PHPT) in India continues to demonstrate a distinct clinical profile compared with Western countries, with several major Indian series documenting a persistently high burden of skeletal disease, severe biochemical derangements, and widespread vitamin D deficiency[1,2].

In one of the earlier large Indian experiences, Jha *et al.*, reported that symptomatic disease with bone involvement continued to dominate the presentation pattern, with 60% of patients experiencing bone pain, 16% presenting with pathological fractures, and 31–40 % demonstrating radiological osteitis fibrosa cystica, highlighting the advanced skeletal burden still seen in India[3].

In the multicentric Indian PHPT Registry reported by Bhadada *et al.*, skeletal involvement remained strikingly common, with 56% of patients presenting with bone pain, 27% with pathological fractures, and 40% demonstrating radiological features of brown tumours. This study emphasised that vitamin D deficiency, delayed diagnosis, and higher preoperative PTH levels contribute significantly to the persistence of severe bone disease in Indian populations[1].

Similarly, in the large contemporary series by Sadacharan *et al.*, comprising 804 patients from South India, musculoskeletal manifestations were reported in 35% of cases, with pathological fractures in 4.5% and brown tumours in 4.1%. Despite a rising proportion of asymptomatic presentations in metropolitan centres, this dataset clearly demonstrates that overt skeletal disease continues to constitute a substantial burden of PHPT in India[4].

International literature contrasts sharply with these observations. Large Western cohorts, including those described by Silverberg and colleagues, show a predominance of asymptomatic disease, with <5% of patients demonstrating overt skeletal manifestations and most cases detected incidentally on routine biochemical screening[5].

These studies reaffirm the distinctive Indian phenotype of PHPT which is mostly characterised by younger age, higher biochemical severity, profound vitamin D deficiency, and a disproportionately high burden of skeletal complications[1,3,6]. Despite delayed diagnosis and florid bone disease, surgical outcomes remain excellent, with consistent biochemical cure and substantial skeletal recovery. These findings underscore the need for earlier detection, routine calcium screening, and timely endocrine evaluation for patients presenting with lytic lesions, fractures, or unexplained bone pain.

4. Material and Methods

This is a retrospective review of four patients with biochemically proven PHPT and skeletal manifestations, treated surgically between [2023–2024] at the Department of Endocrine Surgery, Madras Medical College. Clinical, biochemical, and radiological data were reviewed. All patients underwent parathyroidectomy following localization with ultrasound and sestamibi scans. Follow-up data included serum calcium, Parathyroid Hormone (PTH), Alkaline Phosphatase (ALP) and Dual Energy Xray Absorptiometry (DEXA) Scan.

5. Results (Including Observations)

5.1 Presentation of Cases

CASE 1

A 35-year-old female presented with complaints of inability to walk for the past three months and a swelling over the right leg for one year. She had a history of low backache for one and a half years, insidious in onset and progressive, aggravated on walking and relieved on sitting or lying down. She was initially evaluated elsewhere, where MRI of the whole spine revealed T1-hypointense, T2- hyperintense lytic lesions involving the L2, L3, D5, and C7 vertebrae (Figure 1(A, B)) along with an anterior wedge compression fracture of the

D12 vertebra. Serum electrophoresis showed a normal pattern, ruling out multiple myeloma.

A curettage and bone grafting was performed for a 2.5×2 cm lytic lesion in the middle one-third of the right tibia, and histopathology showed osteoclastic giant cells. The patient was subsequently referred to our centre for further evaluation. At presentation, biochemical investigations revealed severe hypercalcemia with serum calcium of 15.1 mg/dL (8.5–10.5 mg/dL), hypophosphatemia with phosphorus of 1.9 mg/dL (2.4–4.5 mg/dL), markedly elevated alkaline phosphatase of 1948 IU/L (30–120 IU/L), elevated intact PTH of 2956 pg/mL (16–65 pg/mL), and vitamin D deficiency (14 ng/mL). DEXA scan showed severe osteoporosis with T-scores of −5.3 at the lumbar spine, −4.5 at the hip, and −4.2 at the distal radius. Radiographs demonstrated classical skeletal changes of primary hyperparathyroidism, including subperiosteal bone resorption in the radial aspect of middle phalanges (Figure 1(C)), a "salt-and-pepper" appearance of the skull, and brown tumours involving the tibia and fibula (Figure 1(D)). There was no family history of hypercalcaemia or endocrine tumours, and she had no clinical features suggestive of MEN1, MEN2A/2B, or hyperparathyroidism–jaw tumour syndrome.

Localization studies included a neck ultrasound which showed a 2.1×2 cm hypoechoic lesion in the inferior aspect of both thyroid lobes. Tc-99m sestamibi scintigraphy demonstrated tracer uptake corresponding to both inferior poles (Figure 1(E)).

The patient underwent bilateral neck exploration, during which the right inferior parathyroid gland measured 1×1.5 cm and the left inferior gland measured 3×2×1 cm. The left inferior gland was excised and histopathology confirmed a parathyroid adenoma, while the right inferior gland showed normal parathyroid tissue.

Postoperatively, her serum calcium dropped to 8.1 mg/dL and PTH to 27 pg/mL. She developed hungry bone syndrome, requiring intravenous calcium supplementation. At one-year follow-up, the patient had regained full mobility and was able to perform daily activities independently. DEXA scan showed significant improvement in bone mineral density, alkaline phosphatase levels had decreased to 350 IU/L, and serum calcium remained stable at 8.2 mg/dL on oral calcium and vitamin D supplementation.

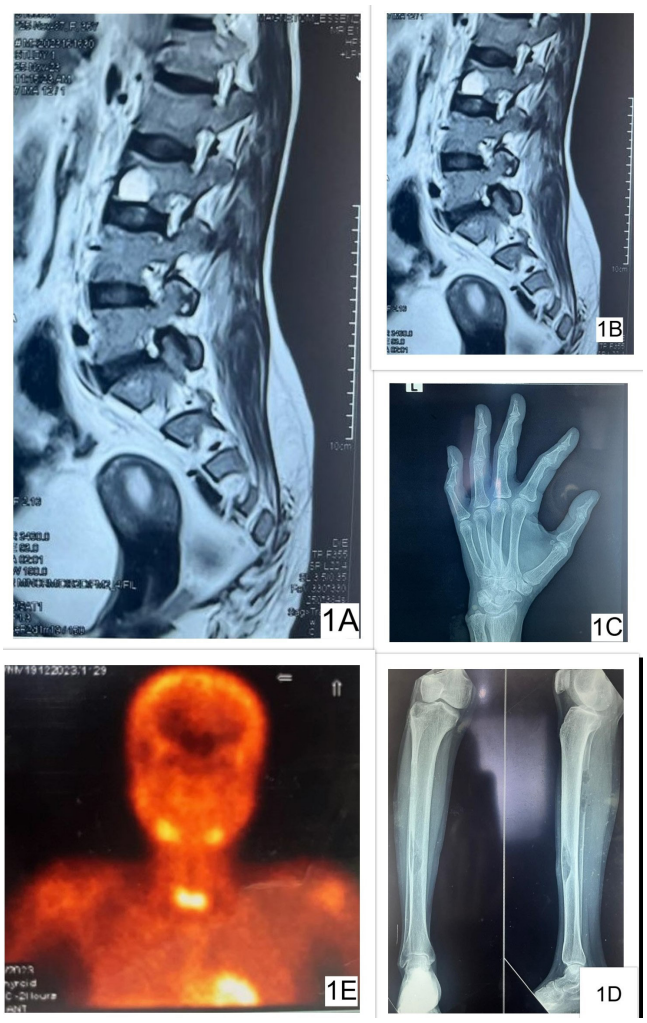

Figure 1. **(A), (B).** Mri spine showing lytic lesions lumbar vertebrae. **(C).** XRAY image showing subperiosteal bone resorption. **(D).** Brown tumour of tibia and fibula. **(E).** TC99 SESTAMIBI showing parathyroid lesion.

CASE 2

A 28 year old female presented with complaints of inability to walk for the past 1 month. She had initially developed left knee pain associated with difficulty walking about three months earlier. She was evaluated elsewhere, where MRI of the left knee showed a solid–cystic lesion in the distal third of the femoral shaft measuring 3.2×2.1×3 cm (Figure 2(A),(B)). To rule out malignancy, a PET-CT was performed, which showed

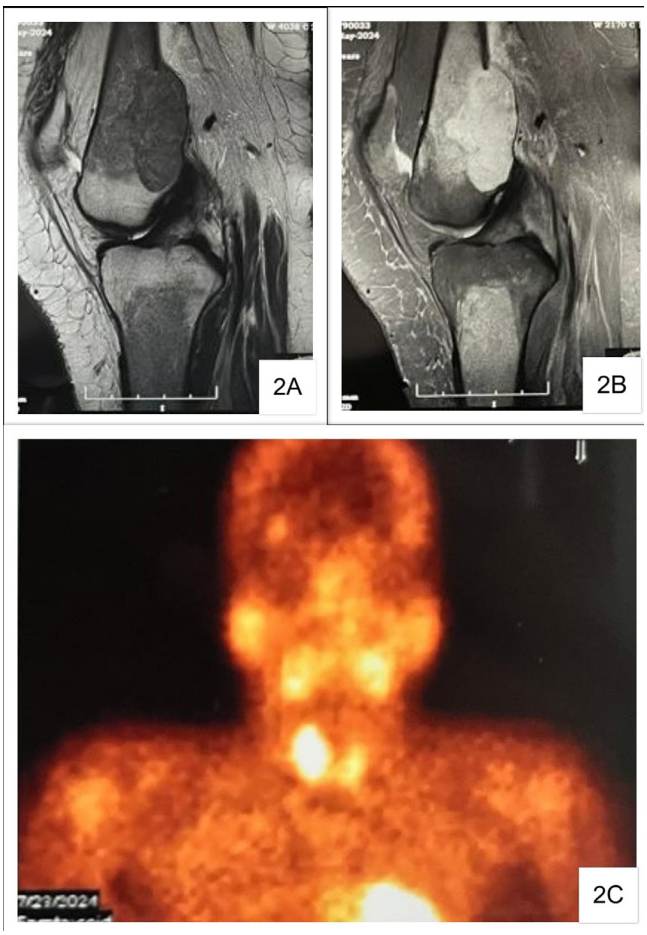

Figure 2. **(A), (B).** MRI knee image showing T1 hypointense and T2 hyperintense solid cystic lesions respectively. **(C).** TC99 SESTAMIBI showing right parathyroid lesion.

additional lytic lesions in the right maxillary sinus, head of the humerus, right third and fifth ribs, both scapulae, and the left tibia. Bone marrow biopsy was negative for hematological malignancy, and biopsy of the left knee lesion showed no evidence of malignancy. As subsequent testing elsewhere showed raised serum calcium levels, she was referred to us for further work-up.

At presentation to our centre, biochemical evaluation revealed moderate hypercalcaemia with serum calcium 12.4 mg/dL (8.5–10.5 mg/dL), phosphorus 2 mg/dL (2.4–4.5 mg/dL), PTH 870 pg/mL (16–65 pg/mL), vitamin D 35.9 ng/mL (30–80 ng/mL), and ALP 600 IU/L (30–120 IU/L). DEXA scan demonstrated osteoporosis with T-scores of −3.8 at the lumbar spine, −2.9 at the right hip, −4.5 at the left hip, and −4.5 at the distal radius.

Usg localised 2.2x1.6x1.2 cm well defined heteroechoeic lesion noted posterior to the superior pole of the right lobe of thyroid. Tc 99m sestamibi : positive for tracer uptake in the lower poles of both lobes of thyroid gland with prominent gland in the right lobe region (Figure 2(C)).

Patient Underwent bilateral neck exploration with right superior (2x1.5x1 cm) and left inferior parathyroidectomy (1x0.8x0.5 cm). Histopathology confirmed a right superior parathyroid adenoma, while the left gland was normal.

Postoperatively, her calcium dropped to 9.5 mg/dL and PTH to 24pg/mL. She developed hungry bone syndrome requiring intravenous calcium supplementation, after which she was discharged on oral calcium.

At one-year follow-up, she was ambulant and performing daily activities independently. DEXA showed improvement to osteopenia, ALP had decreased to 130 IU/L, and serum calcium remained stable at 8.5mg/dL.

CASE 3

A 29-year-old male presented with a two-year history of progressive bony pain, multiple fractures, and inability to walk. He also reported progressive limb deformity and shortening over this period. On examination, he had obvious shortening of the lower limb, rachitic rosary (Figure 3(A)), pectus carinatum, and clinical evidence of multiple healed and active fractures.

Skeletal radiographs showed acro-osteolysis (Figure 3(B), 3(C)), a classical salt-and-pepper appearance of the skull, severe osteopenia, and healed fractures of the ulna (Figure 3(C)) and brown tumour of femur (Figure 3(D)). Biochemical evaluation revealed marked hypercalcaemia (serum calcium 14.9mg/dL), low phosphorus (1.6mg/dL), very high alkaline phosphatase (2175IU/L), profoundly elevated PTH (2456pg/mL), and vitamin D deficiency (9ng/mL).

Localization studies revealed a 3.5×2×3 cm heterogeneous solid lesion with internal calcification on ultrasound, situated adjacent to the superior pole of the left thyroid lobe. Tc-99m sestamibi scintigraphy

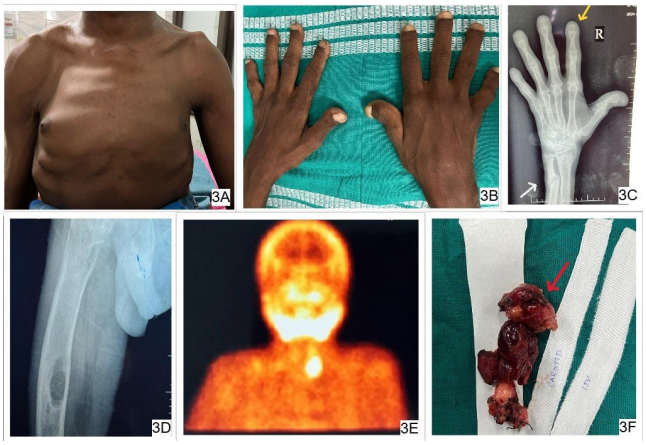

Figure 3. **(A).** Rachitic rosary. **(B), (C).** Acro-osteolysis. **(D).** XRAY of Brown tumour femur; 3E- TC99 SESTAMIBI showing parathyroid lesion**. (F)** Specimen picture.

showed abnormal tracer uptake at the lower pole of the left thyroid lobe(Figure 3(E)).

Given the severity of biochemical derangement, skeletal involvement, and large gland size, parathyroid carcinoma was considered preoperatively. He underwent left superior parathyroidectomy with left hemithyroidectomy and left central compartment clearance (Figure 3(F)). Intraoperatively, the left superior gland was markedly enlarged, measuring 3.5×3×2.5 cm.

Postoperatively, serum calcium fell to 8.2 mg/dL and PTH to 25 pg/mL. He developed hungry bone syndrome, requiring intravenous calcium followed by high-dose oral calcium and phosphorus supplementation.

Histopathology confirmed a left superior parathyroid adenoma with no capsular, vascular, or soft- tissue invasion. The patient was subsequently lost to follow-up.

CASE 4

A 31-year-old female presented with a swelling over the right leg for the past two months. The swelling had been insidious in onset and gradually increased in size, associated with pain and difficulty walking. On examination, there was an ill-defined, bony-hard, tender swelling measuring approximately 2×2 cm over the proximal medial aspect of the tibia.

She had been evaluated elsewhere, where MRI showed multiple lytic lesions with soft-tissue

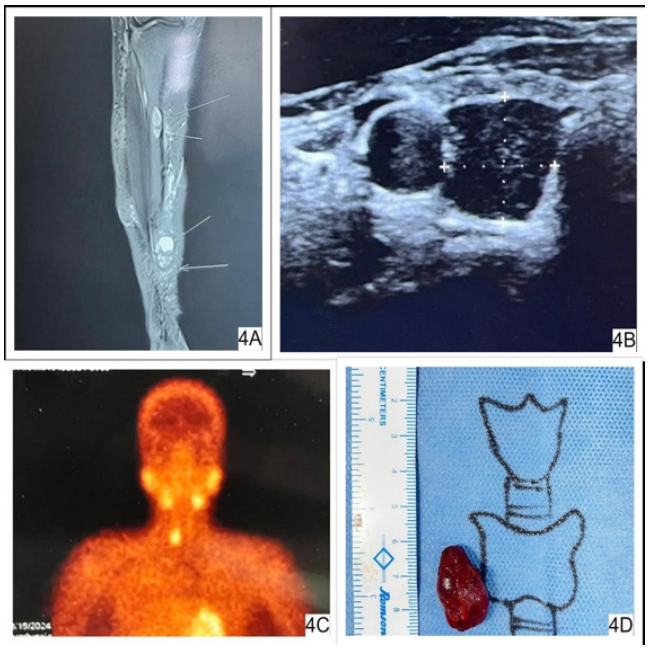

Figure 4. **(A).** MRI showing lytic lesions in the tibia and the fibula. **(B).** USG neck showing parathyroid lesion**. (C).** TC99 SESTAMIBI showing right parathyroid lesion. **(D).** Specimen picture.

involvement involving the upper and lower thirds of the tibia and the fibula (Figure 4(A)). Bone biopsy had been performed, and histopathology demonstrated osteoclastic giant cells. On further testing, her serum calcium was elevated, and she was referred to our centre for definitive management.

At presentation, laboratory evaluation revealed serum calcium 12.3mg/dL, phosphorus 1.8mg/dL, alkaline phosphatase 693IU/L, PTH 1552pg/mL, and vitamin D 8.1ng/mL. DEXA scan showed osteoporosis with T-scores of −3.9 at the lumbar spine, −2.7 at the left hip, −2.6 at the right hip, and −3.1 at the distal radius.

Ultrasound of the neck demonstrated a 1.7×0.9×0.8 cm hypoechoic lesion inferior to the right thyroid lobe (Figure 4(B)). Tc-99m sestamibi scintigraphy showed focal tracer uptake inferior to the right thyroid lobe (Figure 4(C)).

She underwent focused right inferior parathyroidectomy, and the excised gland measured 2.5×2×1 cm (Figure 4(D)). Histopathology confirmed a parathyroid adenoma. Immediate postoperative calcium was 8.4 mg/dL and PTH 20 pg/mL. She

developed hungry bone syndrome and required intravenous calcium supplementation, after which she was transitioned to oral calcium and vitamin D.

At one-year follow-up, her DEXA scan showed normal bone mineral density. Serum calcium was stable at 9 mg/dL, and alkaline phosphatase had improved to 96 IU/L.

6. Discussion

This case series illustrates the severe end of the clinical spectrum of Primary Hyperparathyroidism (PHPT), in which patients present with florid skeletal disease-ostietis fibrosa cystica, brown tumours, pathological fractures and crippling deformity, a pattern that continues to occur in the Indian subcontinent.

In India, PHPT remains a symptomatic disease with more than 90% of the patients presenting with clinical manifestations compared to 70-80 % of assymptomatic disease in the western population[1,3,7].

This striking disparity reflects several interconnected factors: limited routine calcium screening in primary care settings, delayed diagnosis due to under recognition of subtle presenting symptoms, widespread vitamin D deficiency that exacerbates parathyroid hyperplasia, and potentially different genetic predispositions within the Indian population.

The mean age presentation our series was 30.8±3.1 years (28-35 years).This was lesser than with data from the Indian PHPT registry, which reported a mean age of 42±14 years[8]. However, in a study by Bhansali *et al.,* the majority (67%) of patients were less than 40 years old[6].

In the present series (n=4), all patients (4/4, 100%) presented with significant bone pain and functional impairment (difficulty/ inability to walk). Radiological features of subperiosteal resorption and "salt-and-pepper" skull were evident in three of four patients (3/4, 75%), and brown tumours were seen in all patients and pathological fractures were documented in one patient (1/4, 25%). In previously published Indian series musculoskeletal complaints have remained a dominant mode of presentation. Jha et al reported that bone pain and metabolic myopathy were the commonest presentations (≈60%), with pathological fractures in 16% of patients[3]. The study by Bhadada *et al.,* documented bone pain in 56%, pathologic fractures in 27% and Osteitis Fibrosa Cystica (OFC) in 40% of their cohort and Shah *et al.*, found bone pain in 53.4%, OFC in 31.4% and fractures in 34.5% over the period 2005–2010[9], similarly in a Kashmir series of 78 patients reported bony symptoms in 44% with pathological fractures in 10.1% of patients[10]. By contrast, contemporary Western series report overt OFC and brown-tumour presentations in only a small minority (5%) because routine biochemical screening detects disease at an earlier stage[11,12].

In our series, all patients had hypercalcaemia, with a mean serum calcium of 13.7±1.5 mg/dL (12.3-15.1 mg/dl) and a markedly elevated mean PTH of 1960±929 pg/mL(870-2956 pg/ml) . These values are far higher than those reported in major Indian cohorts, where studies by Jha *et al.*, Bhadada *et al.*, and Shah *et al.* have documented mean PTH levels largely within the 300–900 pg/mL range[1,3,9]. The largest contemporary South Indian dataset by Sadacharan *et al.*, analysing 804 patients, reported a mean PTH of 376.5±278.7 pg/mL and mean calcium of 11.4±1.8 mg/dL, with skeletal manifestations in roughly one-third of patients[4]. Contemporary Western screened cohorts demonstrate even lower biochemical severity, with mean PTH levels typically 120–250 pg/mL and serum calcium rarely exceeding 11.0–11.5 mg/dL[5,12].

Vitamin D deficiency is highly prevalent in India, with 70–100 % of the population exhibiting 25-hydroxyvitamin D levels <20 ng/mL, compared with less than 20% in most Western countries[2]. In our series, vitamin D levels ranged from 8.1 to 35.9 ng/mL, with three of four patients demonstrating deficiency (<20 ng/mL), including two with severe deficiency (<10 ng/mL). The coexistence of vitamin D deficiency and PHPT is known to exacerbate skeletal involvement. Low vitamin D enhances PTH secretion and skeletal sensitivity to PTH, increases bone resorption, reduces intestinal calcium absorption, and impairs osteoblastic function during bone remodeling[13]. These mechanisms likely contributed to the marked biochemical severity and florid skeletal disease observed in our patients.

The mean time from symptom onset to definitive diagnosis in our series was 11.8 months (median 10.5 months, range 2–24 months). Although this is

shorter than delays reported in earlier Indian cohorts like Bhansali et al. documenting a median of 3 years (mean 48.9±47.9 months) and Jha *et al.*, reporting a mean delay of 26 months, diagnosis remains considerably slower than in Western screening-detected PHPT, where routine laboratory testing typically enables identification within 6 months and 70–80 % of cases presented incidentally[3-6,14]. Atraumatic fractures and brown tumors may be misinterpreted as malignancies or infectious bone diseases, delaying appropriate intervention illustrated in our case series where each patient initially underwent extensive evaluation, including MRI, PET-CT, bone marrow studies and targeted biopsies, before measurement of serum calcium and PTH. These observations parallel the findings of Lorenz *et al.*, whose multicentre analysis of 135,034 adults with documented hypercalcaemia demonstrated that only 9.7% were ultimately diagnosed with primary hyperparathyroidism, and that delays beyond one year were associated with increased rates of osteoporosis and systemic morbidity[14].

All four patients in this series developed Hungry Bone Syndrome (HBS), attributable to severe skeletal involvement, vitamin D deficiency, and high preoperative bone turnover. Comparable high rates have been reported in Indian cohorts, with Bhadada et al. documenting an incidence of 32% and Bhansali *et al.*, reporting 29%, particularly among patients with marked skeletal disease and vitamin D deficiency[1,6]. Similarly, Witteveen *et al.*, noted that HBS occurred in 25–90 % of patients with radiological evidence of hyperparathyroid bone disease, compared with only 0–6 % in those without skeletal involvement[15].

Surgical outcomes in the present series demonstrated 100% biochemical cure with normalization of calcium and PTH postoperatively, consistent with contemporary Indian surgical series reporting cure rates of 96-99 % in experienced tertiary care centers. Alkaline phosphatase levels, markedly elevated preoperatively due to high-turnover bone disease, declined by 78–86 % at one year, reflecting robust suppression of osteoclastic activity and active skeletal remineralization. These findings parallel observations from Indian cohorts such as Sharma *et al.*, who reported significant BMD gains (8–14 % at spine and hip) within the first year after surgery in symptomatic PHPT, particularly among younger patients and those with severe preoperative osteoporosis[16]. International data are similar, Lu *et al.*, demonstrated substantial one-year BMD recovery, with the lumbar spine showing the highest percentage improvement following parathyroidectomy in patients with moderate to severe PHPT[17].

7. Summary and Conclusion

This case series highlights the persisting burden of advanced skeletal manifestations of primary hyperparathyroidism in India, where delayed diagnosis and vitamin D deficiency continue to increase disease severity. Despite florid osteitis fibrosa cystica, multiple brown tumours, and severe osteoporosis at presentation, all four patients achieved complete biochemical cure following parathyroidectomy with substantial skeletal recovery at one-year follow-up. These findings mirror contemporary Indian and international literature demonstrating that even profoundly symptomatic PHPT exhibits remarkable reversibility once treated. Early recognition of metabolic bone disease, timely analysis of serum calcium and PTH, and prompt surgical referral remain essential to prevent prolonged morbidity and ensure optimal functional recovery in this patient population.

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