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OSTEOPOROSIS TREATMENT PRESCRIPTION IN VERY HIGH-RISK FRACTURE POPULATION: A SINGLE-CENTRE EXPERIENCE

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INTRODUCTION
Osteoporosis is associated with increased fracture risk, leading to significant morbidity and mortality. Despite established clinical guidelines, a substantial gap remains between recommended and actual prescribing practices. Patients at very high fracture risk have poorer outcomes and are recommended to receive anabolic or parenteral antiresorptive therapies. However, access to these treatments is limited in Malaysian government hospitals due to cost constraints.

METHODOLOGY
This retrospective clinical audit evaluated osteoporosis treatment prescriptions among very high-risk patients attending the Osteoporosis Endocrine Clinic at Hospital Queen Elizabeth II from 2020 to 2025. Very high fracture risk was defined according to the American Association of Clinical Endocrinologists criteria, including recent fractures, fractures on therapy, multiple fractures, high fall risk, glucocorticoid use, very high FRAX probability (>30% major osteoporotic fracture or >4.5% hip fracture), or very low bone mineral density (T-score <–3.0).

RESULTS
A total of 120 patients were included, with a median age of 68 years (interquartile range [IQR] 62–73). Most were of Chinese ethnicity (60%, n = 72), with a median body mass index of 22.6 kg/m² (IQR 19.6–26). Secondary osteoporosis was present in 70% of patients, commonly due to aromatase inhibitor use (45%), glucocorticoid-induced osteoporosis (13.3%), thyroid disorders (10.8%), and vitamin D insufficiency (9.2%). Only 12% of patients (n = 14) received anabolic or parenteral antiresorptive therapy, while 88% (n = 106) were treated with oral alendronate. Among those on alendronate, 19.8% (n = 21) developed adverse outcomes, including treatment failure (n = 13), gastrointestinal side effects (n = 6), and atypical femoral fractures (n = 2).

CONCLUSION
The uptake of guideline-recommended therapy in very high-risk osteoporosis patients remains low. A notable proportion of patients treated with oral alendronate experienced adverse outcomes or suboptimal responses. Addressing financial and systemic barriers is crucial to improving access to advanced therapies and optimizing patient outcomes.

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REDEFINING DEFINITIVE THERAPY: PERCUTANEOUS ETHANOL ABLATION FOR PRIMARY HYPERPARATHYROIDISM IN A NONSURGICAL CANDIDATE

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INTRODUCTION
Parathyroidectomy is the definitive treatment for primary hyperparathyroidism (PHPT) due to parathyroid adenoma. However, surgery may be contraindicated in patients with significant comorbidities. Ultrasound-guided percutaneous ethanol ablation (PEA) is a minimally invasive alternative that induces biochemical remission through targeted destruction of hyperfunctioning tissue. We report a case of PHPT successfully managed with PEA in a patient unfit for surgery due to cardiac dysfunction.

CASE
In 2023, a 53-year-old female with stage IB breast carcinoma was found to have persistent hypercalcemia (2.68–3.59 mmol/L) during chemotherapy and post-mastectomy follow-up. Evaluation excluded bone metastases. Biochemical assessment demonstrated elevated intact parathyroid hormone (iPTH) levels 38.6 pmol/L (Reference: 1.6–6.0 pmol/L), hypophosphatemia (0.42–0.80 mmol/L), and elevated alkaline phosphatase (ALP) 215–309 U/L (30–120 U/L)—consistent with PHPT. Tc-99 m sestamibi scintigraphy localized a 1.1 × 1.5 × 1.2 cm hyperfunctioning parathyroid adenoma.

Initial management prioritized oncological therapy, including trastuzumab for 1 year. Hypercalcemia was intermittently controlled with intravenous hydration and zoledronic acid when calcium exceeded 3 mmol/L.

Her disease was complicated with severe osteoporosis (DEXA T-score −3.3) with vertebral fractures, renal impairment requiring cessation of alendronate, and medullary nephrocalcinosis on computed tomography surveillance.

Following completion of cancer therapy, she was evaluated for parathyroidectomy. Preoperative assessment revealed NYHA class II heart failure, with reduced ejection fraction (36%) and severe tricuspid regurgitation attributed to trastuzumab-related cardiomyopathy. Despite optimal medical therapy, she was deemed high-risk for surgery. Cinacalcet failed to achieve sustained calcium control with levels exceeding 3 mmol/L. She was therefore referred for PEA.

Post-procedure, iPTH decreased 80% by Day 5 (54.9–10.6 pmol/L), with sustained normocalcemia (2.25 mmol/L) at 10 days without further need for cinacalcet.

CONCLUSION

This case illustrates that PEA can serve as definitive therapy for PHPT in patients unsuitable for surgery. It provides rapid and sustained biochemical control, while avoiding operative risk, supporting its role in individualized management.

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EXPANDING THE SPECTRUM OF VITAMIN D-DEPENDENT RICKET TYPE 2: DOMINANT-NEGATIVE VDR MUTATION AND POSTPUBERTAL CALCIUM ADAPTATION

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INTRODUCTION

Vitamin D-dependent rickets type 2 (VDDR2) is traditionally defined by biallelic vitamin D receptor (VDR) mutations causing resistance to $1,25(OH)_2D$. We describe a case of the classical VDDR2 phenotype with a single VDR mutation and an interesting physiological adaptation.

CASE

A female diagnosed with rickets at age three presented with a femoral fracture, severe short stature, hypocalcemia (2.1 mmol/L; NR 2.35–2.70), hypophosphatemia (0.8 mmol/L; NR 1.5–2.10), and evidence of renal phosphate wasting (TMP/GFR 0.5 mmol/L; NR 1.5–2.4). Biochemistry showed markedly elevated alkaline phosphatase (1,227 IU/L; NR 50–136) and secondary hyperparathyroidism (20.7 pmol/L; NR 0.8–7.8). Despite hypocalcemia, $1,25(OH)_2D$ was significantly elevated (>450 pmol/L; NR 60–150), consistent with VDDR2. There was no initial family history; however, subsequent evaluation of her mother—prompted by the patient's diagnosis—revealed a similar biochemical profile, along with short stature (142 cm) and a history of multiple fractures. Genetic analysis in both individuals identified a heterozygous missense mutation in exon 10 of the VDR gene, affecting the ligand-binding domain, supporting a pattern of dominant inheritance. The patient was treated with cholecalciferol (1,200 IU daily), calcitriol (5–6 µg daily), and calcium carbonate (2,000 mg daily). Although biochemical responsiveness to therapy was evident, poor adherence resulted in suboptimal metabolic control throughout childhood and adolescence, including a second fracture at age 15 and a final adult height of 124 cm. Notably, calcium and phosphate levels progressively normalized after puberty, with sustained biochemical stability despite the patient omitting the treatment.

CONCLUSION

This case provides two important insights. First, it represents the fourth reported case worldwide demonstrating a dominant-negative effect of a VDR gene mutation, whereby a single mutant receptor interferes with wild-type VDR function. Second, it highlights postpubertal calcium adaptation, in which vitamin D–independent intestinal absorption may restore mineral homeostasis, and disease severity can attenuate over time through adaptive physiological mechanisms.

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GIANT PARATHYROID ADENOMA WITH DELAYED HUNGRY BONE SYNDROME: A CASE REPORT

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INTRODUCTION

Giant parathyroid adenomas (GPAs), defined as lesions >3.5 g, are rare. Their size, biochemical severity, and compressive features often mimic carcinoma, creating diagnostic and surgical challenges.

CASE

A 33-year-old female was incidentally found to have hypercalcemia during evaluation after her newborn developed severe hypocalcemic seizures requiring NICU admission. Maternal calcium was 2.95 mmol/L with hypophosphatemia. Subsequent reviews showed persistent hypercalcemia (3.15–3.3 mmol/L), hypophosphatemia (0.32–0.51 mmol/L), and markedly elevated intact parathyroid hormone (85–96 pmol/L). She had vitamin D deficiency, very high alkaline phosphatase (1,329 U/L), and progressive