Tertiary hyperparathyroidism during high phosphate therapy of familial hypophosphatemic rickets

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We report the development of severe tertiary hyperparathyroidism in three girls treated for familial hypophosphatemic rickets and characterize parathyroid function in vivo and in vitro. All patients had been previously treated with relatively large doses of inorganic phosphorus (125 mm/day) and ergocalciferol or calcitriol for several years and had radiographic evidence of long-standing hyperparathyroidism. Even in the presence of extremely elevated PTH levels, oral phosphate lowered serum calcium levels in vivo and further stimulated PTH secretion. Profound multiglandular parathyroid hyperplasia was found in each patient at surgery. Examination of the secretory characteristics of the excised parathyroid tissue revealed that either relatively high calcium concentrations were generally needed to suppress PTH secretion or PTH secretion was not suppressible. Caution is recommended when relatively large doses of phosphate are used to treat familial hypophosphatemic rickets.